EXHIBIT L

IN THE UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF NEW JERSEY

CHAYA GROSSBAUM and MENCHEN GROSSBAUM, Her Spouse, Individually, and as Guardians ad litem of the Infant, ROSIE GROSSBAUM

Plaintiffs

vs.

GENESIS GENETICS INSTITUTE, LLC, OF THE STATE OF MICHIGAN, MARK R. HUGHES, M.D., NEW YORK UNIVERSITY SCHOOL OF MEDICINE and NEW YORK UNIVERSITY HOSPITALS CENTER, Both Corporations of the State of New York, ABC CORPORATIONS: 1-10 and John Doe

Defendants

_____/

Docket No. 07-CV-359

GARRY CUTTING, M.D.

April 24, 2010

(TITLE PAGE Continued on the Next Page)

Reported by: Linda Lindsey, CSR

10

A. Training to take care of patients, particularly pediatric patients with a variety of different conditions.
Q. Can you define pediatrics for me?
A. Pediatrics is children from neonatal to 18 years of age.
Q. Okay.
A. Sometimes a little longer, in the twenties, depending on exactly the case and condition.
Q. Okay. Um, now is it fair to say your CV, and -- which I have a copy of here and I believe is to the tune of 31 pages?
A. Yes.
Q. Which you produced to -- to -- to counsel?
A. It's the one I sent to Mr. Lewis.
Q. Is that an accurate -- is that -- let's strike that.
Is your CV an accurate -- an accurate description of your medical experience?
A. Yes.
Q. So if there is anything worthwhile in that you did in the medical -- in the medical field it's in here?
A. Sure.
Q. Okay. Now, after your -- your residency in

11

pediatrics you completed a fellowship, correct?
A. Correct.
Q. Okay. We'll -- what did that -- strike that.
What was the concentration of that fellowship?
A. Genetics, medical genetics.
Q. Okay. What does that mean, medical genetics.
A. It's training in the care, diagnosis -- let's start, care and treatments of patients with a variety of genetic disorders.
Q. Can you be -- be more specific? Tell me exactly sort of what that entails?
A. Well, patients whose disease is primarily caused by abnormalities in specific genes.
Q. Okay. Yeah, I'm just trying to get an idea sort of what kind of things did you do? I mean, did you --
A. I would be consulted on patients who were, where the physicians felt that there would be a genetic case because a familial recurrence of children of the same family. So, like CF, two children same family have the disease, so we get consulted for that. We get Sickle Cell Disease. We get consulted for skeletal abnormalities --
Q. Fine.
A. -- a whole variety conditions. And they

12

included both children and adults.
Q. I see. So it's -- it was people already born, obviously, that's what I was trying to focus to. People who are already born --
A. Oh, no, not just that.
Q. Okay.
A. No, we also did consult on prenatal cases as well.
Q. Okay.
A. So we included prenatal counseling. The -- the discussion of things such as teratogens that may affect a pregnancy, genetic conditions that may affect a pregnancy.
In addition to my training, I also trained in clinical laboratory genetics, as well as this is called molecular genetics, which I'm board certified in, and as in biochemical genetics. So that was undertaken during the time that I was training in medical genetics.
Q. So biochemical genetics and clinical genetics?
A. And, well, clinical genetics, biochemical genetics and molecular genetics. The molecular is in the diagnosis of disorders by examination of DNA directly.
Q. Okay. Now -- okay. So, what specific

13

function did you do during your fellowship as it pertains to, um, prenatal involvement with patients?
A. So we would counsel patients who had prenatal conditions. We would see patients who had undiagnosed prenatal conditions where there was concern. We would see women who had advance maternal age, which I'm sorry to say, is beyond 35 years of age who have were at higher risk for children with abnormalities, such as Down Syndrome.
If I'm going too fast, please tell me to slow down.
It would be working with families to tell them about the results of test, such as sider genetics tests or -- or DNA based tests and the results and counseling them on those results. With working with them to make decisions about whether to continue the pregnancy or not to continue the pregnancy, particularly if the pregnancy was affected.
Q. And that was during your fellowship?
A. That was during my fellowship and I continue to do that. That's why I'm carrying the beeper today, if this condition came up today at Johns Hopkins, I would be called for it and I would go in and talk to them.
Q. Okay. Now, as part of your fellowship --

well, we'll take it step by step. Did you, um, extract eggs from patients?
A. No.
Q. Did you implant embryos?
A. No.
Q. Okay. You mentioned that you're board certified, you became board certified after your fellowship?
A. Correct.
Q. What boards are you certified?
A. American Board of Medical Genetics for the two laboratory specialities I told you about.
Q. Um-hum?
A. And then in clinical genetics as well which is done through ABMG and now through the -- the AMA, basically.
Q. Okay.
A. It's a recognized -- it's a recognized speciality now by the AMA.
Q. Okay. Now, in looking at your CV, I see here that you have few positions that you currently hold that you've held for a substantial period of time. Um, and those, specifically, are, we'll go through them one by one.
A. Sure.

Q. You have the Director of Post-Doctoral Training Programs in Medical Genetics at Johns Hopkins University. 1992 to present?
A. (Witness moves head up and down.)
Q. Okay. Well, what's the medical genetics program at Johns Hopkins, what is -- what -- what does that do?
A. It entails the -- again, the medical genetics training program, you're saying, or the entire medical genetics program, I just want to be clear.
Q. Well, the medical genetics program?
A. Program, okay. So the program involves both the care of patients, which is clinical arm, research and disorders that are both genetics, and education. So, the -- this is specific to the education arm, which is teaching new trainees, which it intends to be M.D., M.D. Ph.Ds and Ph.Ds, in the diagnosis, management and treatment of patients with genetic disorders.
Q. Now, when you -- when you, ah, said diagnosis of patients with genetic disorders, what is -- what -- what's -- what's done at this center? I mean, I'm trying to understand some of this program. What kind of patients come to this program? Couples who want to have kids?
A. Yes.

Q. People who are already pregnant?
A. Yes.
Q. I mean, what -- what --
A. Yes, the entire range.
Q. Okay.
A. Prenatal cases all the way through adults.
Q. All right. So, prenatal cases. Explain to me what kind of patients do you get, prenatal patients?
A. Prenatal cases can range to, again, woman who have an advance maternal age, and so we know they have an increased risk with having children with chromosomal disorders. Women who might have or be concerned that they've been exposed because they take certain drugs or have been exposed to alcohol and other things and are concerned about the affect on the fetus.
It could be individuals who are risk for having a monogenic disorder, a single gene disorder, that is could be sickle cell disease, could be beta thalassemia, it could be cystic fibrosis, polycystic kidney disease, Marfan's syndrome; does that give you a general idea or should I continue.
Q. Okay. Are these -- these patients already pregnant when they come to see you?
A. Yes, some are already pregnant and are some considering pregnancy.

Q. Okay. So for the ones that are considering pregnancy, what's your intro -- what's is the role of your -- what's the role of this medical genetics program?
COURT REPORTER: I'm sorry, I didn't understand you.
Q. What's the role of the medical genetics program vis-a-vis their diagnosis and treatment?
A. To advise them, using nondirective counseling about the options that they have.
Q. Well --
A. The options for diagnosis of the disorder, should they be at risk.
Q. Um-hum?
A. Because genetics involves risks. As far as if it's a recessive disorder where it takes a gene from the father and a gene from the mother to get a disorder, the risk one in four. And while that may seem pretty straight forward to a couple considering a pregnancy and having a child with a condition, that takes sometimes some going over to explain really what one in four means.
Q. But besides one in four --
A. Or it could be one in two, it could be -- there -- I don't mean to define recessive, I was --

Q. Okay.
A. -- just giving you examples --
Q. No, I understand.
A. -- that leads me what else you would like to know.
Q. What I'm trying to figure out is -- is, sort of what services do you provide tell besides telling these patients, okay, this is what you are -- this is the category of risk in which -- in which you fall into --
A. Um-hum.
Q. -- and here are your options, what else do you do?
A. Are you asking what else does the program do?
Q. The program.
A. The program, obviously, offers diagnostics during the during the pregnancy to evaluate whether or not the pregnancy is affected. That would include CVS, chorionic villus sampling, which is 12 to 16 weeks, or amniocentesis which is done infrequently these days, and those are done through the program in general, and primarily by our obstetrics gynecology perinatology group, so --
Q. Okay.
A. Or maternal fetal medicine, actually. But our

trainees are trained in that area as well.
Q. Okay.
A. We get exposed to that.
Q. Now, is IVF something that your program offers?
A. We do not -- no -- IVF, invitro fertilization is a program of obstetrics and gynecology. It is not under the institute of genetic medicine.
MR. HAMAD: Okay.
(Knock at door.)
THE WITNESS: Sorry, where were we, I apologize?
MR. HAMAD: Can you read that back?
(WHEREUPON, the requested testimony was read back.

A. Invitro is not a part of the genetics program here. Just like surgery is not under genetics.
Q. So just so we're clear, I think you said something, make sure the record is clear.
Invitro fertilization is under the umbrella of gynecology and under medical --
A. Obstetrics.
Q. -- genetics? Obstetrical, of course.
A. Yeah, right. You're in the same ballpark.

Q. Same area. All right. So now -- okay. So the service you offer would be to counsel the patients about their -- the level of risk that they have, the options that they have available to them, and then to conduct amniocentesis testing or CVS testing after the pregnancy has occurred, fair?
A. Yes.
Q. Okay. All right. Now, you're also the director of DNA laboratory I see here?
A. DNA Diagnostic Laboratory.
Q. Yeah.
A. Correct.
Q. What does that mean DNA -- DNA Diagnostic Laboratory?
A. So we receive samples on patients who are at risk for genetic disorders and we type the DNA to determine whether or not they indeed have the disorder for which the physician is concerned about.
Q. What kind of physicians refer -- refer DNA information to you?
A. Refer samples to us?
Q. Samples, yeah.
A. Okay. So it could be a variety of different physicians. Could be internists, pediatricians, geneticists, basically, the whole gamut.

Q. Ob/gyn's?
A. Ob/gyn.
Q. Reproductive endocrinologists?
A. It could be, yes.
Q. Okay. And what -- what kind of thing -- they send you a sample, what do you do with it?
A. Well, we extract the DNA, and type the DNA.
Q. Okay.
A. That is the simplest answer I can give you, but please I would be happy --
Q. No, that's fine.
A. -- to add more than that. I don't mean to be simplistic more than necessary, so I wasn't sure what you're asking me.
Q. Please as simplistic as you can.
A. Okay.
Q. I'm only a lawyer.
A. Okay. No, no.
Q. Which doesn't mean very much these days.
All right. So, you receive genetic samples, you type out the DNA and then you send something to the physicians?
A. Yes.
Q. Okay. What kind of genetic disorders do you test for?

22

A. There's about 40 genetic disorders we test for currently in the laboratory, ranges all the way from disorders involving abnormalities in the blood, sickle cell disease, the globin disease that I was mentioning, thalassemia, certainly includes cystic fibrosis. A variety of what we call skeletal dysplasias, abnormalities of bone growth.

I'm just trying to think of all the categories. Abnormalities of the genes that contribute to the growth of the aorta, Marfan's syndrome, TTGF beta receptors -- I won't need get more specific. I'd be happy to give you the Web site of the lab and you can take a look at the range of disorders for which we provide diagnostic.

If I could simplify, it's basically, primarily conditions in which one gene causes the disease, when there's a defect in the gene. That's pretty much what we do.

Q. But just so I understand your role in the overall, I guess, continuum of care for patients --

A. Um-hum.

Q. -- is it fair to say that patient goes reproductive endocrinologist office, they're counseled that they might be at risk for something, a sample is taken, they send it to your laboratory, you do the

23

testing, you send the result back to the reproductive endocrinologist, is that where your laboratory fits in?

A. Yes, yes, that's where our lab could fit in.

Q. Okay. That makes sense to me?

A. Okay.

Q. That's how simple you have to be. It's -- it's sad.

A. All right.

Q. And you've -- you've been doing that since 1994 and still currently do that?

A. Yes.

Q. Okay. There's a lot things that I see in your -- in your that say 1992 or 1994, whatever the year may be to the present. What I would like you to do -- do as we move, are continuing along, position by position, tell me how much of your time is spent doing each of these various or holding down all these various positions.

A. Sure.

Q. So, the first position that we covered was Director of the Post-Doctoral Training Programs in Medical Genetics?

A. Ten percent.

Q. Ten percent?

A. These are all documented by the way, I can

24

give you the documentary reporting that's required by the NIH and required by my hospital.

Q. I was hoping for estimates. That's good.

A. That's why I can give you specific answers.

Q. Awesome.

All right. The director of the DNA Diagnostic Laboratory?

A. Fifteen percent.

Q. Okay. Professor of Pediatrics and Medicine?

A. That's primarily my research that would take about 70 percent.

Q. And Director of Genetic Residency Programs?

A. Well, that's actually folded into that ten percent, it's the same kind of thing, it's all my education component.

Q. That actually, it concluded in 2008.

A. Yes, that's right, I did turn that over to one of my junior trainees, who's now the director of the residency program.

Q. Okay.

A. So, that was folded initially, but became a second responsibility so I turned it over to somebody else.

Q. You know what, I'm going to throw a little wrench here, because I think I -- I should have asked

25

you these questions a little give way.

A. Okay.

Q. Between 2003 and 2005 --

A. Yes.

Q. -- with the numbers that you already gave me, the percentage breakdowns stay the same?

A. Yes.

Q. Okay. And for the remainder of this deposition when I ask about, sort of what it is that you do or whether your functions, your laboratory functions throughout your various positions, I'm referring to that period of time, 2003 and 2005?

A. Fair enough.

Q. Okay. Now, we also have, you said in 2003 the genetic residency program would have been folded into the ten percent the post-doctoral training program?

A. Yes.

Q. Okay. Then we have the Director of Cystic Fibrosis Genotyping Center?

A. That is part of my research activities, it would folded into the 70 percent.

Q. Okay. All right. You have five percent left?

A. Yes.

Q. What's that for?

A. That's primarily to account for the times

right now that I take off.
Q. Okay. Make senses.
A. We won't -- we won't go into exactly how this is allocated, but there is a -- it's not clearly -- it's a long five percent the best I can tell you.
Q. That's all right.
A. But if you want to get down to specific hours, I would happy to give them to you.
Q. No, no that's fine -- that's fine. So, now let's discuss the, I guess what takes the bulk of your time which is being Professor of Pediatrics and Medicine. You indicate -- you indicated to me that that's primarily your research position?
A. Um-hum. Right.
Q. Or function?
A. That's correct. Based on my functions.
Q. What is it exactly that this, being a professor of pediatrics and medicine, and doing all -- what -- what do you do exactly? What do you research?
A. I run a laboratory of about 15 people.
Q. Okay.
A. Investigate the various molecular genetics of cystic fibrosis. Make it even simpler genetics and cystic fibrosis.
Q. Um-hum. When you say investigates the

genetic -- mole -- molecular component of genetics of cystic fibrosis, what does that mean exactly?
A. Well, the different changes in the gene that contributes to the disease, how it changes the functions of the protein, how the function of the protein changes the properties of the cells, and how that changes the way that the lungs work, for example, and why patients get disease. And that is all aimed to increasing our understanding of this condition so that we develop therapist therapies.
Q. Again, I'm just trying to understand sort of -- sort of to see where you fit in the continuum of care, where your laboratory fits in the continuum of care for patients.
Who would -- how would it be that you get, I guess, samples for this laboratory?
A. Which one?
Q. The -- well, you mention here that the pediatrics and medicine, I mean, that's -- or that is all folded into one?
A. No. So for the research laboratory, I -- I conduct a series of research projects that are approved by internal review board of John Hopkins.
(Knock at door.)
THE WITNESS: So, you were asking?

MR. HAMAD: So, where were we?
THE WITNESS: Yeah, where were we?
(WHEREUPON, the last answer was read back.)
THE WITNESS: So these are all studies that involve research in which we recruit patients from, actually, all over the United States with cystic fibrosis and diseases related to cystic fibrosis, and we have been studying their genetics causes, and I have been doing that for a good 25 years.
Q. So is it fair to say you don't -- and this is in 2003 and 2005 and even now, you don't refer sample from patients to laboratories and get results from laboratories, it's actually the opposite. People send you samples and you send them out results, fair?
A. But I do both.
Q. Well --
A. So, let me explain.
Q. Yeah.
A. So, when I'm on service or I see patients in the clinic, I send samples to other places as well.
Q. Okay.
A. Because my laboratory does not do -- the DNA Diagnostic Laboratory does not do every test available. So I'm involved in sending samples to other commercial and academic laboratories for genetic testing.

Q. What percentage of your time do you spend in the clinical portion where you would actually see patients and send samples to other places?
A. It's -- it's registered as five percent of my time.
Q. Okay.
A. And that's the figure I will give you.
Q. All right. So, and that's true -- so, in that five percent of your time between 2003 and 2005, you would sometimes see patients that required testing of some sort that your laboratory didn't do?
A. That's correct.
Q. And that you -- in those situations you would send those ah, ah, tests out to someone?
A. That's correct.
Q. But your laboratory does the CF testing, fair?
A. For CF our own laboratory would be, generally, the place, yes.
Q. Okay. Now, um, this clinical experience, five percent, that you spend five percent of your time doing, um -- well, strike that.
Your clinical experience, I mean, what exactly do you do? What kind of patients do you see?
A. Sure. So I have a clinic once a month. I have on one Monday coming up, so I'll see eight to ten

mutations, fair?
A. Yeah, disorders and -- yes, correct.
Q. Is it fair to say that a substantial number of the articles that you've -- articles slash book chapters that you've published deal with the issue of the various types of genetic mutations that cause CF?
A. Yes.
Q. Would you consider yourself an expert in the area of the, specific area of the genetic mutations that cause CF?
A. Yes.
Q. Okay. How much of your time, Doctor, would you say out of, I mean, again, you have the 70 percent, the 15 percent for diagnostic laboratory the, um, the post-doctoral training program, medical genetics, how much of all that effort and time is spent working and concentrating on testing and researching and, um, I guess, well, testing and researching the issue of the specific gene mutations that cause CF?
A. Out of all the activities?
Q. Yeah.
A. Probably 20 or 30 percent.
Q. Okay. Now, you mentioned you had four depositions before today as an expert witness?
A. I think it's four.

Q. You think it's four. No, problem.
A. I think it's four.
Q. Let's -- let's take them one -- let's just start with this. How many were on behalf plaintiffs?
A. Um, I think three, one on defendant.
Q. Okay. All right. Step back --
A. Obviously, the other way around.
Q. Let's step back for a second. I know you said you sat for four depositions?
A. Yeah.
Q. Estimated four?
A. I estimate.
Q. How many times have you asked to review a case for lawyer and render an opinion?
A. Maybe ten times.
Q. Ten times. How many reports have you authored?
A. Um, maybe eight reports.
Q. Okay. Out of those ten times you were asked to review a case how many plaintiffs asked -- how many were for plaintiff's attorneys?
A. I'm going -- this is an estimate.
Q. Um-hum.
A. I'm guessing, might be just a little more than half.

Q. Okay. Estimating, fair.
A. Yeah, I'm estimating.
Q. The word guessing legally is -- is --
A. Well, I said -- estimating is a little bit -- so guessing, no idea what it really --
Q. Yeah -- estimate --
A. I think --
COURT REPORTER: Please stop. One at a time.
MR. HAMAD: That was my fault.
THE WITNESS: Equally my fault. I'm going to say maybe five or something must have been for the plaintiff.
Q. All right.
A. It might have been six I, you know, I don't keep a record of this.
Q. No problem. And out of the reports you authored, how many were for the plaintiff, if you remember? If you can estimate for me?
A. No, didn't we say eight for --
Q. Of the reports?
A. -- of the reports, I believe.
Q. Okay. All right.
A. That's what I thinking. Yeah, you asked me of the reports.
Q. All right.

A. But I think that's what it was.
Q. Now, what percentage of your time is spent providing expert opinion to attorneys?
A. One percent or less.
Q. Do you charge a fee, obviously?
A. Yes.
Q. For service?
A. I do.
Q. What is your fee?
A. Four hundred dollars an hour.
Q. For what?
A. For providing expert evaluation of a case.
Q. Does that include review, drafting the report, depositions, trials?
A. Deposition personal appearance are $800 an hour.
Q. Okay. And trial testimony?
A. Same price. Any personal appearance I charge $800. I do not charge for the travel time, that's the fairest way I think I can do it and keep it close.
Q. Okay. Now, if we can -- what states -- strike that.
The cases in which you authored reports what states have those cases been venued?
A. One was in New Jersey. I can't recall what --

38

I don't remember -- one was in New Jersey. The other was in a northern midwest state, I can't recall exactly.
Q. They all blend together.
A. Well --
Q. Michigan, Minnesota, who cares?
A. I have to say that -- I dispose of the records afterwards, I don't keep the records.
Q. Yes.
A. And I don't actually keep a memory. And so much, so little of my time is spent doing this.
Q. Okay.
A. I apologize.
Q. That's --
A. It should be a matter of public record if you want to look it up.
Q. That's fine.
A. But I do -- I do remember New Jersey where the defendant -- for the defendant in that case. And there was a case, I know in midwest, I recall. I'm sorry, I can't recall the others right now.
Q. No problem. Okay. Now the case in New Jersey, um, who were the -- which attorney retained your services?
A. I don't remember.

39

MR. STEIN: You might try to find out how long ago we're talking about here.
MR. HAMAD: Well, we're going do get there, Lou.
MR. LEUCHTMAN: While we're all chiming in, the ten cases that you reviewed were over what period of time?
THE WITNESS: A good ten years or so.
MR. LEUCHTMAN: Thank you.
Q. I was going to ask him.
A. Well, now you know.
Q. See that. Per year how many reports or cases do you review?
A. Let's see, ten by ten, about one a year, I guess.
Q. So one a year. Okay.
A. Or so. Sometimes they get clustered might be two and then a couple I'll go a years with none.
Q. Have you ever provided trial testimony?
A. No.
Q. Okay. Now, I know you don't remember the venue of these cases or maybe even the lawyers that hired you but, um, I'd like to you to tell me about the facts of each case?
A. Okay.

40

Q. Um, would any -- were any of the dealing with -- were any of the cases that you provided expert reports did they deal with the issue of PGD, IVF.
A. A couple, yes.
Q. A couple. Okay. And what were the facts of those cases?
MR. LEUCHTMAN: And why don't you do them separate PGD and IVF.
Q. Were they -- okay.
A. They were PGD.
Q. PGD?
A. Yeah.
Q. Okay.
A. One was a case involving, actually, RGI in Chicago, with a misdiagnosis. I don't actually recall the -- I don't recall the -- the actual condition they were doing. Um, another, um, PGD, there was -- there's been two other CF cases where they were misdiagnoses on, well, one for sure I can recall is a misdiagnoses based on a prenatal diagnosis, not a -- not a PGD, sorry. And then one other one involves a PGD case, for CF, I believe.
Q. When you say prenatal misdiagnoses versus PGD, what -- what do you mean?
A. Well, I mean, I think it was a contention of

41

an error done, actually, after the child -- it was in utero test that -- it was a on the fetus done at 12 weeks, CVS based test by the laboratory and the laboratory had not properly reported the information.
And there's one, yeah, the other one Florida I remember. You see now you get me to think for a second I can remember the other case. The another one is in Florida, again a very straight forward case with CF where the couple came for counseling and the doctor, well, erroneously counselled the family.
Q. Okay. And that involved --
A. No.
Q. -- PGD?
A. That -- well, it involved -- no, it didn't involve PGD. No, it involved, two of those cases the birth of the child with CF, those cases.
Q. Okay. So we have a Florida, one CF case, no PGD?
A. Yes. That one I recall.
Q. We have one prenatal CVS test regarding CF; where was that case, do you recall?
A. Which one?
Q. The one with prenatal for CVS testing that --
A. Well, that might have been the state of Maryland.

62

the first sentence here of your -- of your -- of your second paragraph -- I'll -- I'll restate it. I'll pause afterwards and hear your judgment.

I have formed opinions that there are two areas where Genesis Genetics and the NYU IVF Clinic failed to offer reasonable level of care?

A. Yeah.

Q. Is it fair to say that in the -- in this second paragraph you talk about the -- the -- the first alleged failure, and in the third paragraph of your report you talk about the second alleged failure, which is for Genesis Genetics, fair?

A. Yes.

Q. Okay. All right. Now, offer a reasonable level of care, what does that mean, Doctor, what did you mean by that?

A. What I would expect a group or any other organization offering the same care to -- to offer as if they were offering the same service somewhere else in the United States.

Q. Can you be a little more specific? Explain to me what's -- what's a reasonable level of care? What -- what is reasonable level?

A. Reasonable level of care, well, I will say it again. What I would expect another similar clinic or

63

similar operation or similar university or commercial outfit to offer if this was a service they were providing. It's an equivalency concept.

Q. Equivalency --

A. As in equal to.

Q. Equal --

A. Is the care provided New York University up to the levels I would expect of care to be provided at some other institution.

Q. Okay. And you're basing that upon your own institution?

A. More than that.

Q. Okay. What?

A. Well, my understanding from reading the literature, from going to meetings, to talking with colleagues, to traveling extensively --

Q. Okay.

A. -- to talking with -- there is a certain level of care when it comes down to these areas that would be reasonable to expect.

Q. Okay. I'm just trying -- so is -- is what you're telling me is, if I understand you correctly, that from your experience this is what you do, this is the kind of level -- strike that.

From your experience, I guess, in the field of

64

CF and genetic testing, that this is what Johns Hopkins does, and that you would expect other institutions to do what Johns Hopkins does?

MR. STEIN: That's not an accurate restatement.

MR. HAMAD: I'm -- I'm asking him, Lew, with all due respect if that's fair. If it's not fair he will tell me.

A. I'm not holding them to Johns Hopkins. I'm holding it to what I think is a reasonable level of care at other institutions. And I base that on, not only my experience here at Johns Hopkins, but my interaction with other colleagues in the field, with what's in the literature and what I see at meetings.

Q. Okay. Now, um, you have here a criticism -- strike that.

You say the first referencing to the criticism you have of NYU or you opinion against NYU in this case is, quotation mark here. "The first is in the counseling of the Grossbaums regarding alternatives for embryo transfer after it was discovered that the embryos recommended by Genesis Genetics were not suitable for transfer." Did I read that accurately?

A. Oh, sorry, it's the second sentence.

Q. Yeah.

65

A. Couldn't find it.

Q. Okay. Now, discovered that the embryos or the recommended embryos were not suitable for transfer, what do you mean by that?

A. One of the two embryos was not suitable for transfer.

Q. Okay. What -- what do you mean? Can you explain to me what not suitable for transfer means?

A. This is what was described in the records, not suitable for transfer.

Q. Okay.

A. However deemed by them, it was not suitable for transfer.

Q. Okay. So, you have the records in front of you to show me what you're talking about. One second. You don't need that thing. What you're saying is, avoid the hassle.

If I understand you correctly, you're saying your -- your understanding is that NYU determined that one of the embryos Dr. Hughes recommended was not suitable for transfer for some reason?

A. Precisely what I said.

Q. Okay. Now, who discovered that?

A. From what I understand the IVF lab at NYU.

Q. Okay. And what did they base their -- this

66

judgment that this embryo is not a suitable one?
A. Based on the -- a predicted viability or morphology of the embryo itself.
Q. Okay.
A. So whatever it is, it didn't pass their grading scale.
Q. Do you know what their grading scale is?
A. No.
Q. Do you know what look for, what they test for?
A. No. I'm not an embryologist. Not my area of expertise.
Q. Okay. Only -- only embryologist test embryos to see their progress?
A. It is the area of the embryologist to be an expert in that area.
Q. Can reproductive endocrinologists do that as well?
A. Yes, I believe they're also, reproductive endocrinologists are involved in IVF, yes.
Q. Okay. Is that part of the IVF umbrella, reproductive endocrinologists and -- and embryologists, OB/GYNs, is that --
A. Yes.
Q. -- generally who kind of --
A. Yeah, that's the way I view it. If you're

67

describing as such.
Q. Yeah.
A. And IVF unit would be within OB/GYN and reproductive medicine that would be the area of expertise.
Q. Okay. It would include what specialities exactly, as far as your understanding?
A. Specialities?
Q. Yeah, what specialities --
A. OB/GYN, maternal/fetal medicine, reproductive endocrinology.
Q. Embryology?
A. Embryology, thank you.
Q. Do those all have certifications, those specialists?
A. I believe they do.
Q. Okay. They got to pass boards?
A. I believe they do.
Q. Okay. Can you tell me anything about the process of tracking the embryos after -- in the laboratory by the embryologist?
A. No.
Q. Okay. All right. Now, you reference an allele dropout --
COURT REPORTER: A what dropout?

68

MR. HAMAD: Allele, A L L E L E dropout, is a well established source of error in preimplantation genetic diagnosis.
A. Um-hum.
Q. And then you go on to indicate from the deposition of Dr. Licciardi it was apparent that he was not aware of this potential cause for error?
A. Um-hum.
Q. Is that a yes?
A. Yes. Sorry.
Q. That's fine. She just has to write it down. Um, can you show me where -- strike that. Do you have doctor -- you do have Dr. Licciardi's deposition in front of you?
A. Yes. Sure.
Q. In your -- in your -- can you show me where he -- what portion of deposition he indicates that he does not -- he is not aware of the allele dropout?
A. Fully Aware the consequences.
Q. Sorry.
A. Fully aware of so maybe that -- I think I know where you're going.
Q. I think you -- I think what your sentence says here it's--
A. Yes.

69

Q. Deposition of Dr. Licciardi, it was apparent that he was not aware of this potential cause of error, what does that mean, Doctor?
A. Give me a second.
Q. Sure. Sorry.
A. You asked me to look at records. Hang on.
Q. Yes. The deposition is what he referenced, Doctor, sorry.
MR. LEUCHTMAN: Since I have something of an outline if you could provide us with a page and line I'd appreciate it.
THE WITNESS: Yeah, I'm trying to find it. Okay. All right. So, we're on page 50.
Q. Okay.
A. I think this must have been your examination of him. And it says, line seven: We see as with regard to sample two, T only mean. What does T only mean? Answer from Dr. Licciardi, I don't know.
Regarding 3 there is no amplification, he corrects himself, no regarding 4 and 7 the letter G appears, what does G mean? I don't know.
In regard to 8 there is a G/T, what does that mean? I don't know. And regarding now, we move over to Call, what's the meaning of Call? And then we go through this, so here's indicating.

70

Now, he says, page 51, at the top, page of one. Okay. So, sample 2 is possibly affected, and it says ADO paternal. And I assume allele -- ADO means allele dropout. Yes. What is the mechanism of allele dropout? When the test is performed and you don't get your answer, the feeling is you're unable to test one of the alleles.

And then we go, no molecular signal, I take it that that's not an embryo that can be successfully used for fertilization; is that correct? Oh, sorry, that's going on beyond the ADO part.

Q. Okay. So what I'm asking, Doctor, is that -- have you read for me the only basis that's the basis for your statement that Dr. Licciardi does not know that allele dropout is a potential cause of error?

A. I don't believe -- well, he's -- my opinion he doesn't understand the principal of allele dropout nor does he understand the results of this genetic test.

Q. All right. Is it fair to say, based upon what you just read now, I mean, if we could reason together you and I for a second, that while he may not understand the -- strike that.

It is fair to say in your -- in your report what you're trying to say is, if I understand you correctly, that Dr. Licciardi is aware that allele

71

dropout is a risk, but he may not understand the exact mechanisms of how -- of how allele dropout occur, fair?

MR. STEIN: I object to the form --

MR. HAMAD: Fair enough. Well, if he understands it.

MR. STEIN: -- of what he's trying to say.

MR. HAMAD: Noted.

Q. Now, Doctor, you can answer it? Is that a fair understanding?

A. Say it again, please? Could you repeat --

Q. I'll rephrase it.

A. I don't mean to be cute.

Q. No, it's fine?

A. To see what you're asking me.

Q. It's fine. It's fine. My job is to make the questions as fathomable, somewhat at least, and Lew's job to teach me how to be letter lawyer?

A. Is he getting there?

MR. STEIN: Here's learning.

THE WITNESS: I'm sorry. We're an hour-and-a-half in.

MR. LEUCHTMAN: The rest of us are just spectators to this dynamic process.

THE WITNESS: Okay. Go ahead, please, I'm sorry.

72

Q. Is it a fair reading of the sentence in your report which reads as follows, "From deposition of Dr. Licciardi, it was apparent that he was not aware of the potential cause error." To say that -- what you're saying is Dr. Licciardi does not know the specific mechanism by which allele dropout occurs, fair?

A. It is more than that.

Q. It's more than that?

A. Not only to does he not understand the mechanism, he doesn't understand the implications, and how it may apply to much more than just that one sample.

Q. Okay. And your base -- and have you shown me in -- in the deposition your basis -- strike that.

Have you -- have you pointed out to me in Dr. Licciardi's deposition the basis for your conclusion that he does not full -- he is not fully aware of the potential cause of error?

A. Well, he -- I just indicated, he doesn't understand the basis of nucleic acid Calls here. If he doesn't understand that then he will not understand not only the mechanism, but the implications of ADO for each of those embryos that were tested, not just number two.

Q. I understand. But my point being, Doctor, you

73

showed me the base -- strike that.

You have -- you have an opinion here in this sentence in your the report that says, Dr. Licciardi apparently doesn't understand ADO, right? You have that opinion here?

A. He does not understand --

Q. You have the -- the basis --

A. -- beyond the mechanism.

Q. Yeah.

A. His action, also, he made a decision as well and that's transfer that other embryo.

Q. I understand. But --

A. And had he don't -- okay. I'm not going to say anything more.

Q. But, Doctor, your sentence says this: From the deposition of Dr. Licciardi --

A. I understand that sentence.

Q. -- it was apparent he was not aware of this cause of error. I'm just asking you, have you pointed -- pointed to me the areas of the deposition where you believe -- where -- where you're referencing in your report in that sentence you pointed those out to me, right?

A. Um-hum.

Q. Yes?

74

A. Yes. This is one source of my opinion.
Q. Fair enough. In the deposition?
A. One source is in the deposition.
Q. Okay.
A. Yeah.
Q. All right. And you can -- those -- those two sources, do you have page 50 and page 51 includes those -- the basis for that conclusion?
A. That's correct, from the deposition.
Q. All right. Fair. What's the other source, Doctor?
A. Um, the choice of transferring embryo number seven.
Q. Okay. Now, you indicated there was no -- no documentation of what was said during the counseling session between Dr. Licciardi and the Grossbaums regarding the risk of potential sources of error; do you see that in your report?
A. Yes.
Q. Okay. Um, what documentation would you -- would there be? Strike that.
What documentation would you be looking for?
A. Documentation of a conversation or whatever counseling session took place that would be in writing, would be part of the medical record that would indicate

75

that there was a presentation of the information and the risks involved in switching an embryo.
And let me be specific with what I mean by switching. If I can find now. This is what we got, so this is his report. This is his report.
Q. Okay.
A. So, this was recommended okay for transfer. This was recommended okay for transfer. (Indicating.) Where does it say that embryo number 7 is okay for transfer here?
Does Dr. Licciardi, did they contact Dr. Hughes? Did he document the context to find out if that was okay for transfer if he didn't understand the results of this test?
Did he -- did he put any documentation that he had discussed that this was not said to be okay for transfer with the family?
It's like changing an operation in the middle and saying, well, I did but I -- I by word of mouth we talked, but I'm not going to document this.
In this day and age a physician knows that any change in the protocol, when information is provided here, and he's relying on this he says completely, which is what says in his deposition, then where has he documented that he made a change of plans?

76

Q. Now, is there conclusion that's reached by Genesis Genetics as it pertains to embryo number 7 in that -- in that --
A. Yeah, carrier -- carrier at worst.
Q. What does that mean? What does carrier at worst mean?
A. The opinion of Genesis Genetics --
Q. Doctor, I'm just asking what does carrier at worst mean?
A. What is meant by this?
Q. Yes. Carrier at worst, just by itself?
A. The -- the idea -- yeah, yeah, I'm getting --
Q. No, I understand.
A. Give me a second here.
Q. Just want to see your notes, actually.
A. Go ahead. Go ahead, please take a look.
Carrier at worst, at least it's written by the thing would mean, the worse thing this could be is carrier.
Q. Okay.
A. I think it is obvious.
Q. Doctor -- okay. I'm going to ask you about this note, Call here, do you see where it says Call, this column here? (Indicating.)
A. Yes.

77

Q. Column that says Call, is that fair to say that that's the conclusion?
A. Yes.
Q. Okay. And they're conclusions for each of the embryos and the control that were tested?
A. Say, I'm sorry? I'm -- I'm after an hour-and-a-half drifting in and out.
Q. Sorry. Now, those samples that were tested, correct?
A. Yes.
Q. We have here 15 samples some of which are not actually embryos?
A. Actually, there were ten embryos tested --
Q. I understand.
A. -- and the parental samples.
Q. I understand, but that's my point. There is 15 samples or at least 15 -- we see --
A. Well, they're numbered up to 15 --
Q. Exactly.
A. -- there's actually ten samples?
Q. I understand. Now, if you looking at them right here you see that there's, look in the Call column, conclusions regarding the status of these ten various samples, right?
A. Um-hum.

102

the tests available these families would go without the tests.

So in order to ensure the lab is accurate they undergo proficiency testing for every single test, clinical test in the laboratory. That involves taking samples that are provided by the College of American Pathology, that's standardized tests or doing an inter lab exchange, so if nobody else is -- if that test not included in the CAP proficiency test you actually share sample with another laboratory doing the same test.

If there's no other laboratory in the world, and we have couple of tests are our own, that are not doing the same tests, then what we do is we take a sample that we had previously tested, we mask it and we run it through laboratory again, so the technician, staff and the interpreters don't know that it's actually a previously tested sample.

This is done for every single test we have every six months. It is required, not only by Johns Hopkins but by CAP.

Q. And what is CAP?

A. That's the College of American Pathologist who oversee a number of laboratories, not just genetic testing laboratories, pathology testing laboratories, hematology, metabolics and so forth.

103

All the -- whenever your blood sample is sent out, most often in a hospital laboratory it's overseen by regulation -- CAP, and -- and there are some others, CAP is one of the most common regulatory bodies.

Q. Do you agree that as a general proposition in the healing arts, whether it be the aspect of healing arts that you are or the practice of medicine or any other such endeavor, that generally speaking one significant index for the proficiency of the practitioner or lab or the institution is the frequency with which it performs a given procedure?

A. No. I don't agree. There are people that perform procedures over and over again and do it poorly. They don't have in place proper QA, QC programs, these have been detected. A hospital here is doing thousands of blood samples, all of them wrong.

There's forensic laboratories found to be doing thousands of samples, same type, over and over again, errors were made, individuals were incarcerated because of errors.

So just because you do something over and over again doesn't mean you're doing it accurately. You might actually do it over and over again and get the same result, that's precision, it doesn't mean it's accurate.

104

Q. Well, all other things being equal, is the quantity of procedure being done a hallmark, not the the only hallmark of proficiency at doing a procedure?

A. I will say, again, I -- it doesn't matter if you do something 100 times or a thousand times wrong, it's not any different than doing it once right.

Q. If an IVF clinic performs 25 IVF cycles in one year, is it less likely to be qualified at preferring cutting edge care than one that does many, many more?

A. I can't say, I'm not --

Q. All right.

A. -- expert in IVF laboratories. So, I've -- I've already indicated I'm not -- I will talk about DNA labs but not -- I could cite to the areas where I have expertise and be more careful.

Q. Now, have you -- strike that.

There are some newer technologies coming along that are purported to be exceptionally good for looking at -- seeing defects in one cell and also looking at chromosomes, such as SNP, chips and microbead technologies, are you using those in your lab?

A. No.

Q. Why not?

A. We don't have applications.

Q. Is that something that a PGD lab would do that

105

you would not? When that's what you mean by not having application for?

A. No, my -- the DNA diagnostic clinical laboratory doesn't currently have applications where we use those kinds of apps, currently.

Q. Well, what applications would cause you to use them?

A. If we chose to test for a variety of common variance that contribute to Type II diabetes in the population, for example. We test for, primarily single gene disorders with just one or few variance contribute to the disease. The technologies are available for those, the highly robust and there are -- there's no reason to change them at the current time.

Q. Over the years of complex testing have there been diagnostic mistakes in your laboratory?

A. That I'm aware of from our internal proficiency testing and follow-up testing, because we do that to see if we make errors, we contact clients and so forth, I'm not aware of any errors that we have -- there's only one I can think of which actually did not -- was picked up before it actually went out as a final report. There were a a couple of others we picked up in process because we have QC programs, quality control programs.

114

A. We -- we attempt to work always through the physician because the physician and/or genetic counselor are the one who contacts us.
Q. Yeah?
A. No, I'm done.
Q. How is PGD different from what you routinely do in your lab?
A. If we're talking about blastomere biopsy which the removal of one cell or two, depending on which lab you do and how you do it, it involves a much smaller amount of DNA and it requires exquisite levels of, um, testing ahead of time to assure that you get an accurate diagnosis, because unlike in the case, as you have been indicating, that when you get a large amount of DNA from a CVS sample or from a blood sample of a patient we're getting six picograms of DNA on average from one cell.
Q. This is in PGD?
A. PGD, sorry.
Q. All right.
A. So the test and the importance of being accurate and having plenty of additional assays there to be sure you achieve accuracy is absolutely paramount in PGD because it is the more challenging area of genetic diagnosis currently available.

115

Q. PGD is?
A. Yes, by fair.
Q. Now, is the sample use in PGD, if you know, different from the samples you use in your lab?
A. Yes, because they're based on blastomere, which is single cell from an embryo. CVS is actually a sample of the fetal portion of the placenta. Amniocentesis is actually cells shed by the embryo that is present in the amnionic fluid that is drawn when a needle is placed into the --
Q. Now, if a cultured sample from a cytogenics lab or other patient tissue samples has one or two milligrams in it, and the PGD sample has two picograms, then would you agree that there's one billion times less DNA in the PGD sample?
A. Has six picograms but, yes, it has quite a bit less.
Q. On the order of of a billion times less in PGD?
A. Well, what's important is the genome equivalence not the amount of DNA.
Q. But I'm -- I'm just asking you as to DNA?
A. Okay. Fine.
Q. Are we agreed?
A. We're agreed.

116

Q. Okay. Would it be correct to say that PGD pushes molecular DNA testing to the limit because it tests, first of all, one cell, which is the smallest unit of life?
A. Yes.
Q. And for one gene which is smallest unit of inheritance?
A. That's not correct, but anyway, yes, we'll say --
Q. For one gene?
A. We'll stick with it.
Q. All right.
A. I'm being a scientist type, let me shut up, yes.
Q. And more often times a change on one A or T or G or C, character in 3.3 billion DNA letters of the human genome?
A. Yes.
Q. Is there any lab testing you're aware of that's anymore complicated than PGD?
A. Yes.
Q. And what would that be?
A. There's lots assays that involve protein biology which involve use of antibodies which have actually quite tricky characteristics in order to

117

properly identify the proteins, and these proteins can actually predict whether you're going to get cancer, you're going to get -- you're actually have metastasis of cancer.
In fact, there's a whole range of these assays available, and they're quite complex to carry out and do, and they acquire exquisite levels of controls and inter assay controls and so forth. So, yes, there are more complicated ones than this.
DNA is a very simple thing to work with, the simplest chemical we probably work with.
Q. Now, your laboratory does not at present do PGD?
MR. STEIN: He said that.
MR. LEUCHTMAN: Yes, I know, it's foundational.
A. Yeah, at present.
Q. Was there a time when you did that?
A. Yes, yes.
Q. When?
A. Over the past couple of years we've done two cases for CF.
Q. Other than that have you ever been involved in PGD?
A. No.

118

Q. Specifically, you're not involved in PGD directly in the year 2004?
A. 2004, no, I don't think we – no, no.
Q. Okay. Well, then let's clarify when you say you have done two --
A. Two cases were done after 2004 and before today.
Q. All right. Well, you said the last couple of years?
A. No, no, you're right. You're absolutely right.
Q. Okay.
A. I don't mean --
Q. Well, let's narrow that down because it's important, I think, in this case.
A. Yes.
Q. When were these two cases --
A. I would say --
Q. -- PGD cases for CF in the past couple of years?
A. -- in the past probably couple of years, I'm doing it again, sorry. Probably more than 12 months ago, but not more than three years ago.
Q. Okay.
A. Is about when I recall them. They were about

119

one and a half to two years apart, if I recall correctly.
Q. So, as -- as best you can recall as we sit here today, the first one you did was in perhaps 2007?
A. Yes. Something like that.
Q. And no earlier than that?
A. Yes, about that.
Q. All right. And you were not involved in any form or fashion directly in PGD in 2004?
A. In delivering the test. We certainly were spending time doing assays to develop the tests, yes.
Q. But you weren't doing PGD testing?
A. No.
Q. No, you were not?
A. No, you're correct.
Q. The two -- the two PGD cases you've done in last three year were for cystic fibrosis?
A. Correct.
Q. And what means did you do to do those tests?
A. A single cell from a cleavage embryo, so it was eight cells, we took out one cell, in the same way this method was done and did a diagnostic to see it.
Q. When you say the same way this method was done --
A. Okay.

120

Q. -- the same thing that Hughes did in this case?
A. Yes, well, as was done by both NYU and Hughes.
Q. Okay.
MR. HAMAD: I'm sorry. I may have missed something. The last couple of years? What -- when was the timing of these tests?
MR. STEIN: Between 12 months and three, he said. Twelve months and years?
THE WITNESS: Yeah.
MR. LEUCHTMAN: Yeah.
MR. HAMAD: Go ahead. I'm sorry.
MR. LEUCHTMAN: And no earlier than 2007.
THE WITNESS: Yeah.
Q. Well, did -- did the two tests that you did three years or less ago involve genomic markers?
A. Yes.
Q. Did they involve polar body biopsy?
A. No.
Q. Do you agree there are many safeguards that need to be put into place in PGD testing over and above, for labs that do not do it?
MR. STEIN: Over and above what?
MR. LEUCHTMAN: Well, for labs that do testing, such as -- as Dr. Cutting does. There are

121

safeguards that exist for PGD that do not exist in -- in the diagnostic labs -- in diagnostic labs such as the one that Dr. Cutting operates.
MR. STEIN: I object to form of the question, but he can answer it.
A. Um, we did offer PGD within our lab using the same safeguards that we use for the other tests. It's just as important to develop the right assays for a CVS test to make sure it's not contaminated with maternal cells, which is a maternal cell test we do using linked marker, and we've being doing for a decade to be sure that we don't have contamination that leads to a misdiagnosis. So we take the same level of care with every test we do.
There are different technical challenges in executing the test which require additional or other approaches and so forth, PGD is one that requires some additional tests, and there are other ones that we do in our labs that do require, also, additional marker tests, but I give you the example, we've been using linked markers, genomic markers for over ten years for maternal contamination CVS. To prevent --
Q. For maternal contamination?
A. Yes. That's finding mother's cells in with the baby's cells when you do the sampling of the

182

mentioned it myself.
Q. Now, I don't know if I asked you for this, it is getting late in day. Does your lab at any time do polar body --
A. No, no, no, we don't.
Q. You do you agree that even today polar body biopsy is not a mainstream approach?
MR. STEIN: Mainstream, objection to form.
Q. In the United States, granted that you're not going to be all that familiar with the PGD community?
MR. STEIN: Approach to -- objection.
A. Certain conditions people would argue that it's a good application. I -- I -- it's a difficult question to answer. It -- it his comments aside. Could you ask that again and give it to me precisely.
Q. Okay.
A. See if I ask answer it as best I can.
Q. Yes. And I'll add -- well, first of all do you agree that even today polar body biopsy is not a mainstream approach in the PGD community in the United States?
MR. STEIN: Same objection.
Q. Okay. Noted. Go ahead and answer the question.
A. So, the single cell analysis it is an option

183

but it is the major one, no, it is an option so it is there.
Q. Okay. Do you expect to offer any opinions in this case as to the existence of a causal relationship between the action or failure to act by any of the defendants and the misdiagnosis or failure that occurred?
A. Yes.
Q. All right. Do you expect to offer such opinion as to Dr. Hughes, just yes or no?
A. Yes.
Q. Do you agree that because of the many variables involved in PGD, the cause or causes of PGD misdiagnosis are always difficult to pin down?
A. Say that again.
Q. Do you agree because of the many variables involved in PGD, and we've discussed some of them, potential mislabeling or --
A. Right.
Q. -- transaction position of things --
A. Yes.
Q. -- et cetera, et cetera? The cause or causes of PGD misdiagnoses are always difficult to pin down?
A. Always difficult to pin down. I don't agree with that.

184

Q. Generally difficult?
A. No, I don't agree.
Q. Can each of the following things be possible causes of failure or misdiagnosis of PGD mosaicism?
MR. STEIN: Are you talking in this case or abstractly?
A. In any case?
Q. Can they be possible causes of failure or misdiagnosis in PGD, generally?
A. Can they be?
Q. Yes.
A. Yes, mosaicism can be which leads to ADO which you need to detect.
Q. All right. What is mosaicism?
A. Mosaicism is the fact that the embryo -- the cells of the embryo contain different numbers of chromosomes or difficult portions of the chromosomes. Instead of them having an entire compliment of 46 chromosomes they my make additional amounts or subset and some of the cells have it and some of them don't, so it's mosaic. So ...
Q. As of 2004 was it possible to predict mosaicism with any degree of medical probability?
A. Mosaicism had been recognized as a condition seen in egg cell embryos and in animals decades ago,

185

and humans also.
Q. Okay.
A. It was known --
Q. It was know that it occurs --
A. Okay.
Q. -- in any given case --
A. Yes.
Q. -- was it possibly to predict mosaicism?
A. Predict whether mosaicism was there or appreciate that it could be there?
Q. Predict in a given case that it could --
A. Well, sure if you aggregated the entire embryo and test each one of them, yeah, you could show --
Q. But that isn't done?
A. Well, you could take two of the cells and that was shown when you take two cells that there was mosaicism. So this I was known by 2004.
Q. As PGD was practiced was it -- was it possible to predict mosaicism more likely than not?
A. It was reasonable to expect that it would -- it could potentially complicate the case, yes. That's about as far as I can go. I don't know where you're going with this question.
Q. Was mosaicism possible, not necessarily a probable or the probable but --

186

A. A possible, yes.
Q. -- the cause of the failure of misdiagnosis in this case?
A. The failure to detect a mosaicism or to exclude a mosaicism was a problem in this case.
Q. Was there a mosaicism in this case?
A. Don't know, he didn't run the linked markers, did he, so we won't know. Had he run them we would have had a better idea if there was mosaicism here.
Q. Well, I asked you before was it possible in 2004 to predict mosaicism with any degree of medical probability?
A. I see your question now, I apologize, you're correct. If you were using linked markers, yes it does help telling you whether there was mosaicism?
Q. It helps but I mean --
A. Yes, it actually can tell you. All -- all markers fail that you run, I'm saying you could assume that that cell might indeed be mosaic, it's missing that region.
Q. If you look at the entire universe of possible causes or failure, causes of failure or misdiagnosis in this case, can you without speculating ascribe a percentage of that possibility to mosaicism?
A. If we see mosaicism ADO rates, by -- it's be

187

recorded by different labs at different numbers but, at least, six percent is the number I've seen bandied around. Others claim that it can be in high depending on how they look at it, as 25 percent. Detected as allele dropout or dropout of several markers, which interprets mosaicism.
I'm cagey because you're asking me in absolutes and the problem is one infers from --
Q. Well, I'm asking can you ascribe a percentage without --
A. Yes.
Q. -- engaging in speculation?
A. Sorry, yeah --
Q. So --
A. So those numbers --
COURT REPORTER: One at a time, please.
THE WITNESS: I apologize. So I apologize. These are the numbers I've seen published they range for mosaicism in cases.
Q. How does DNA contamination occur, particularly six years ago in 2004?
A. It can occur due to errant cells of the person doing the biopsy in the embryo getting introduced into the -- into the tube where the single cell from the embryo is. It can be introduced once the tube is then

188

opened again in laboratory doing the diagnostic testing. It can be present reagents. It can present in a number of places.
Q. Was DNA contamination a possible cause of the failure or misdiagnosis in this case?
A. Um, it is -- contamination is a possible cause, yes.
Q. Well, if you look at the entire universe of possible causes of failure or misdiagnoses in this case, can you without speculating ascribe a percentage of that possibility to DNA contamination?
A. No.
Q. Do you believe it's more likely than not there was allele dropout in this case?
A. Yes, I do.
Q. What's the basis for that?
A. We have an affected child.
Q. Do you agree based upon the literature most misdiagnosis in PGD setting is due to intercourse or unprotected sex?
A. Say again?
Q. Do you agree that based upon the literature that most misdiagnosis in a PGD setting is due to pregnancy with --
A. Most, no. No, no.

189

Q. Is it a --
A. Is it a known problem?
Q. Yes.
A. In the universe of things.
Q. You don't agree that most misdiagnosis or failure is due to it?
A. Again, you're talking about every case out there. You're talking about ones where there are compounds, where their dominants or whatever else, no, if I were to include all the types of test I'm aware, parents who just paid quite a bit of money to have a test, who've gone through a lot, that they're going to go ahead and have unprotected intercourse.
It is a known complication, but it is a pretty rare one, that if you're going to go through all those things that you're going to have unprotected intercourse and have a child affected when you've gone through that, so. I don't think it is.
Was it a mainstream precedent, it was certainly recorded in a few cases, but that's not my understanding that it is common. And logic would dictate from what I just said, that it -- it follows for those reasons that unprotected intercourse for a couple going through this amount of extra work to have a child would actually go ahead do that.

190

Q. Do you agree that the failure rate of condoms is higher, generally, speaking than the failure rate of PGD?
A. I'm not -- I can't comment on that.
(Laughing.)
Q. You don't know?
A. I'm not expert in condom manufacture.
Q. All right. All right.
A. It's a interesting question.
Q. Well, you haven't read the depositions of the Grossbaums, so I mean, it is funny to --
A. Keep going. I apologize. I apologize, please keep going.
MR. STEIN: You think that in the deposition of the Grossbaums they said they had unprotected sex?
MR. HAMAD: Let's move along.
THE WITNESS: He has questions, and I want to make sure that I get your questions.
MR. STEIN: These questions are absurd.
MR. LEUCHTMAN: No, they're not, Lew.
MR. STEIN: Yes, they are.
Q. Were you informed as to anything about whether the Grossbaums had sex shortly before the implantation or after --
A. I have no idea.

191

Q. -- the implantation, whether or not protected?
A. I have no idea.
Q. You have no idea?
A. No.
Q. Is that an important factor to look at in assessing whether there was pregnancy with a non-implanted embryo?
A. Whether there was a pregnancy with a non-implanted embryo. I mean, if I don't know if they did or didn't know for sure -- I don't know what their status is, so I can't answer that question.
Q. So in order to say whether or not what the Grossbaums did or didn't do at -- at the times indicated is a factor in this case, you'd have to have more information than you have now?
A. Say again. To know -- if they said they did or didn't --
Q. Well --
A. -- how are you going to prove they did or didn't have sex.
Q. Well, they might say something about it but you don't --
A. No, I don't know.
Q. -- you didn't read their deposition?
A. If there is information you're withholding

192

from me --
Q. That I'm withholding from you? Did you ask Mr. Stein for the depositions?
A. Well, I had -- fair enough. Fair enough.
Q. Did you?
A. No, I didn't. Fair enough. I did not start with this couple who had a child with cystic fibrosis, a life living disorder, had actually spent money to go and have this test expect that they had done so, that's why I have not asked for the records, nor suspected that this would be a likely cause of misdiagnosis.
Q. Can you say more probably than not any one of the possible causes of failure or misdiagnoses in this case was indeed the cause of failure or misdiagnosis?
A. Unprotected intercourse was that cause.
Q. No, no. Can you say more probably than not that any one of the possible causes of the failure or misdiagnosis in this case was indeed more likely than not the because of the failure or misdiagnosis?
A. The incorrect diagnosis of the embryos which were specifically implanted back into the mother is the most likely cause of this pregnancy being affected.
Q. Well, of all the possible causes, it may be your opinion that it's the most likely, are you saying more likely than not the implantation of an affected

193

embryo caused the bad result in this case?
A. Yes, that's my opinion.
Q. Have you read the transcript of the deposition of Mark Hughes?
A. Yes.
Q. Were there any factual errors in Dr. Hughes's deposition?
MR. STEIN: I object to form of the question.
A. Um, hang on. Hang on.
MR. STEIN: You want to give him the deposition and read now and to tell you if he finds any factual errors.
MR. HAMAD: I think you just asked him, when you read it did something jump off at you.
MR. LEUCHTMAN: Yeah. Sitting here today --
THE WITNESS: Fair enough. I just -- I just sat for more than four hours, give me a second.
Q. Okay. I'm not responsible for all four of those hours.
A. Yeah, I know. Fair enough. Fair enough. Let me see the deposition and see if I have any highlights there and then I can actually take a look at it. Here it is. All right.
Okay. So let's start out. So, first of all he says that, on page number 17. He says, well, has

198

you expect to voice?
A. Regarding the two keys opinions we're discussing with the two of you guys, yeah.
MR. LEUCHTMAN: All right. Let me -- let me confer with Mr. Dickson for a couple of minutes and then I'm done.
(WHEREUPON, a discussion was held off-the-record.)
Q. I just have a couple of questions. Just very quickly what ICSI?
A. ICSI, it is a set of plasmic sperm injection. It is a taking of the sperm head and --
MR. STEIN: Help her out.
THE WITNESS: ICSI, intracytoplasmic sperm injection, ICSI. Yes, so it's a direct injection of the sperm into the oocyte to do -- to do -- to further abrogate mother nature's role in this whole thing, where you're actually manipulating, sticking the sperm head right into the egg.
Q. And it's basically one sperm per egg?
A. That's the idea.
Q. Can contamination occur in ICSI process? Can the sperm be contaminated, for example, by tissue from the prostate or otherwise?
A. Can -- can the sperm be contaminated? Well,

199

I've heard of cases where two sperms are injected. Um, so I guess you could contaminate it, yes. In the realm of all possibilities, yes.
Q. Do you know whether or not ICSI was performed in this case?
A. I don't know for sure, I assume it was. I -- I would have to look it up.
Q. Finally, do you have an opinion as to whether the failure of Genesis Genetics and/or Mark Hughes to do testing with genetic markers, in and of itself is more likely than not the cause of a bad result in this case?
A. More likely than not.
Q. Do you have an opinion as to whether -- strike that.
MR. LEUCHTMAN: That's all I have.
EXAMINATION BY MR. HAMAD:
Q. Doctor, I have a few questions for you.
A. Do you want to switch places.
Q. I'm going the stand right next to you because you and I got to do a little math together.
All right. So here we have Cutting 1, right?
A. Yes.
Q. This is your report, you and I have talked about before?

200

A. Yeah.
Q. Now, for number 7 here there's no amplification here under CF 10?
A. Yes.
Q. All right. Now when you have no amplification of the genetic -- of the genetic mutation, what are the chances that that genetic mutation is going to carry an afflicted --
A. Fifty percent.
Q. Fifty percent?
A. Yeah.
Q. Okay. Now, what if -- what's the chance here when you have going across the line for CF 11, you have a G, right?
A. Um-hum.
Q. For CF 11. What are the -- what are the odds of allele dropout, Doctor, that that G right there which means --
A. Could be as high as 25 percent.
Q. Could be, and as low as what?
A. It could be zero.
Q. Could be zero. So between zero and 25 percent. So in order to determine, Doctor, what the risk that this -- that this conclusion is wrong, carrier at worst, you have to multiple both, correct?

201

A. Yes, that's one way to do it.
Q. So, 50 percent, Doctor, right?
A. Um-hum.
Q. Times zero to 2.25, correct?
A. Um-hum.
Q. Okay. Meaning that this could be -- this has a zero percent chance to a 12 percent, I guess?
A. That's one way to calculate it, I guess.
Q. Okay. In your -- in your opinion, touh obviously, this would be wrong?
A. Yes. But one other thing that you're not factoring in is the fact that there was amplification, may mean the cell was actually in poor situation and this actually is more likely to be in error than even 25 percent.
Q. But do you know -- let me ask you a question?
A. Do I know that? I'm sorry, we've got to slow down.
Q. Yes. But my question to you, do you know what the condition of cell number 7 was?
A. No, all I can detect --
Q. Okay.
A. -- he wasn't able to amplify.
Q. All right. Okay. So that's the information you have.

A. Do you see there's a further nuance that maybe I'm not conveying to you here very carefully or thoughtfully. And I can show you that.

So what you say is you know there's a G and a T, so yes I agree, it's carrying the mother's mutation, but -- but you have got both alleles. You know that you were able to amplify both, therefore it's somewhat more likely that both genes were in there and there wasn't a complete loss of the gene.

For all you for 7, it may have been neither gene might have been in there. It just happened.

So it's a matter of interpretation of the results as stated here. I'm --

Q. Okay.

A. -- taking into account, so it can be --

Q. Can we just do this, Doctor, you know, I have this, you know, I want to get done with this today. Really, I think I'm probably done here.

Doctor, look at number 2, sample 2?

A. Yes.

Q. What does it say there on the very tend at the top?

A. Yes, ADO paternal.

Q. Okay. It says, possibly affected, right?

A. Yes.

Q. ADO paternal?

A. Right. But that's actually, he feels detected ADO because he finds the T.

Q. Does it say that anywhere else?

A. No.

Q. Okay.

A. I mean, you have read my --

Q. Doctor, have you told me everything?

A. Everyone knows that, and it's disclosed, that he's read all my notes, my evaluation of this.

Q. Of course. We're going to mark those as exhibits it will be attached to the deposition?

A. Fair enough.

Q. So, Doctor, just briefly, the same questions you were asked at the end of counsel's questioning of you. You've told me -- have you apprised me today of your opinions that you expect to testify about at trial?

MR. STEIN: Objection to form. Go ahead. Answer the question.

A. Yes.

MR. HAMAD: That's it, no further questions.

(CUTTING Exhibit Nos. 2 through 14 were marked for identification.)

(Deposition was concluded at 5:24 p.m.)

State of Maryland
County of Baltimore, to wit:

I, Linda Lindsey, CSR, a Notary Public of the State of Maryland, County of Baltimore, do hereby certify that the within-named witness personally appeared before me at the time and place herein set out, and after having been duly sworn by me, according to law, was examined by counsel.

I further certify that the examination was recorded stenographically by me and this transcript is a true record of the proceedings.

I further certify that I am not of counsel to any of the parties, nor in any way interested in the outcome of this action.

As witness my hand and notarial seal this 30th day of April, 2010.

_____

Linda Lindsey
Notary Public

My Commission Expires:
December 21, 2011

this letter to Mr. Stein --

A Yes.

Q -- what did you interpret your purpose in this litigation to be? What -- what -- what purpose were you writing this letter for?

A A purpose of offering an expert opinion on diagnostic procedures in PGD.

Q So you -- fair to say, you reviewed yourself at that time as an objective observer who is just going to give Mr. --

A Yes.

Q -- to tell Mr. Stein how it is? Is that your role?

A Yes. That's what I believe an expert is supposed to be doing.

Q So what difference does it make, Doctor, as to what purpose a lawyer wants to use the information or the opinions you give him for? The opinions shouldn't change, right?

MR. STEIN: You are arguing with the witness.

MR. HAMAD: Okay. I'll rephrase it. You are right.

Q Doctor, do you believe that your opinion as a medical/legal expert should change based upon what

said here, could it have changed it if expected under any appropriately done evaluations, under any appropriately done evaluations. I was considering "under any appropriately done." And appropriately done would have been with DNA markers. That's how I understood the question.

Q Would appropriately done now -- appropriately done evaluation also including -- included the Genetics mutations?

A The appropriately done analysis in 2004 when this was done should have been done with genetic markers.

Q Okay. All right.

(The reporter asked for clarification.)

A Genetic or DNA. Let's call them DNA -- DNA markers. That would have been the appropriately done. That's how I understood the question. That was the focus of my answer.

Q Buck Stromme? You know Buck Stromme --

A Yes.

Q -- M.D?, Right? Dr. Stromme?

A Yes.

Q You are familiar with him?

A I know who he is.

Q You know who he is?

Q Do me a favor. Just answer my questions and don't cut me off. Okay.

A I apologize.

Q All right. Now, how many reports like that one you are looking at right now, the one that's dated July 19th, 2004, have you produced to IVF centers who have parents that they want to implant with embryos based upon your report?

A Based on PGD alone and not other cases of prenatal diagnosis where the same type techniques are used are you saying?

Q Yeah.

A PDG, two cases. In cases of other similar diagnosis --

Q I didn't ask you the other similar --

A Hundreds of cases. Hundreds if not thousands.

Q Okay. All right.

A We developed the markers for these kinds of tests for this gene.

Q You develop the marker, but you don't do the actual testing?

A Oh, we -- not for PGD, no.

Q Oh, oky. All right. So you don't do the testing that is done by Dr. Hughes, right?

A So I -- do you mean I can't interpret it?

Q I'm not -- just answer my questions.

A Okay.

Q You don't do the testing done by Dr. Hughes, right?

A Yes, I do. I do a PGD. I have done a couple of cases with CF using -- using linked markers as --

Q Do you know how many cases Dr. Hughes does?

A Doesn't matter.

Q Do you know how many cases Dr. Stromme did in his career?

A Probably hundreds.

Q And you did two?

A Yeah. So what?

Q Okay. Dr. Hughes may have done thousands?

A I have no idea how many he has done.

Q And you did two? Dr. Kang Pu, do you know how many he has done?

A No.

Q Okay. And you feel comfortable disagreeing with all these --

A Absolutely. Absolutely.

MR. HAMAD: I have no further question. Thank you.

EXHIBIT M

Curriculum Vitae

Name:	Frederick Licciardi, MD
Address:	712 Beechcrest Drive River Vale, New Jersey 07675
Date of Birth:	February 15, 1960
Marital Status:	Wife: Josephine Children: Julia, Joseph, Michael

Education:

1978-1982	Rutgers University, Rutgers College New Brunswick, NJ Major: Microbiology
1982-1986	UMDNJ-Rutgers Medical School Degree: MD

Postdoctoral Training:

Residency:

1986-1990	**Resident** in Obstetrics and Gynecology Chairman: James L. Breen, MD Saint Barnabas Medical Center, Livingston, NJ

Fellowship:

1990-1992	**Fellowship** in Reproductive Endocrinology Director: Zev Rosenwaks, MD Cornell University Medical College New York, NY

Licensure and Certification:

1987	**Diplomat** of the National Board of Medical Examiners New Jersey License Registration No. 50317 New York License Registration No. 182351
1995	**Board Certified**, American College of Obstetrics and Gynecology
1998	**Board Certified**, Reproductive Endocrinology

Faculty Appointment:

Associate Professor, Director of Oocyte Donation
Department of Obstetrics and Gynecology
New York University School Of Medicine
New York, NY

Hospital Committees:

2005	Hospital Safety Committee
2006– 2007	Resident Education Committee

Professional Affiliations:

Board Member: The New York Society of Reproductive Medicine - 2002 – present

Executive Council: The Society of Assisted Reproductive Technologies – 2003 - 2005

Fellow: American College of Obstetrics and Gynecology
American Fertility Society

Courses and Seminars:

1992	Course Faculty: Micromanipulation of Gametes and Embryos. Cornell University Medical College
1999 - 2005	Course Faculty: The Art of Donor Oocytes Charleston, South Carolina
2000	Course Co-Director: The Art of Donor Oocytes 2000. Charleston, South Carolina
2001	Course Faculty: Annual Review of Pre-implantation Embryology Cancun, Mexico
2002	ASRM Seminar: A National Donor registry Seattle, Washington
2006	Course Director: In Vitro Fertilization and Infertility New York University
2007	Course Director: Advanced Workshop in In Vitro Fertilization New York Academy of Medicine

2007 Course Faculty: Ovarian Cancer Survivors Course, Gynecologic Cancer Foundation, New York, New York

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21. **Licciardi F**, Berkeley AS, Noyes N, Krey L, Grifo JA. A two vs. three-embryo transfer: The oocyte donation model. Accepted for the American Society of Reproductive Medicine annual meeting, October 2000.

22. Nasseri A, Mukerjeet T, Berkeley AS, **Licciardi F**, Krey L, Copperman A. Predictors of discordant outcome in paired ovum recipients. American Society of Reproductive Medicine, October 2000.

23. **Licciardi F**, Sanghui A, Lawson F, Krey L. Crinone vaginal gel vs. intra-muscular progesterone: Clinical and endocrinologic differences. American Society of Reproductive Medicine, Orlando, 2001.

24. Styne A, Krey L, Kwiatkowski A, **Licciardi F**, Noyes N. Pregnancy outcome and complications in women age 40 or older undergoing in vitro fertilization with autologous and donor oocytes. American Society of Reproductive Medicine, Seattle 2002.

25. Kump L, Jansen V, Baek K, Lee S, Schiffman M, **Licciardi F**. Extensive screening of oocyte donors is nonetheless a worthwhile process. American Society of Reproductive Medicine, Seattle, 2002.

26. **Licciardi F**, Kump L, Berkeley A, Noyes N, Grifo J, Krey L. Sharing oocyte donors makes more sense scientifically, clinically and financially. American Society of Reproductive Medicine, Seattle, 2002.

27. Kump L, Jansen V, Baek K, Styne A, **Licciardi F**. The importance of genetic screening for oocyte donors. American Society of Reproductive Medicine, Seattle, 2002.

28. Montoya A, **Licciardi F**, Krey L. Estradiol: oocyte ratio, an early predictor of reduced ovarian reserve. American Society of Reproductive Medicine, Seattle, 2002.

29. Kump L, Lee S, Schiffman M, Baek K, Styne A, **Licciardi F**. Psychologic screening of prospective oocyte donors. American Society of Reproductive Medicine, Seattle, 2002.

30. Nasseri A, Berkeley A, **Licciardi F**, Krey L, Terzano E, Grifo J. Comparison of ectopic pregnancy rates between patients undergoing embryo transfer on day 3 vs. day 5. American Society of Reproductive Medicine, Seattle, 2002.

Abstracts cont'd:

31. McCaffrey C, Berkeley A, Grifo J, Kump L., **Licciardi F**, Noyes N. Offspring Gender Ratios Differ between Day 3 and Day 5 Embryo Transfer. American Society of Reproductive Medicine, San Antonio, 2003.

32. Fruhman G, Krey L, **Licciardi F**. The Prognosis for Patients with a Cancelled IVF Cycle. American Society of Reproductive Medicine, San Antonio, 2003.

33. Kump L, **Licciardi F**, Krey L, Noyes N, Grifo J, Berkeley A. An Oocyte Donor's Willingness to Donate- Does the Recipient's Lifestyle Make a Difference? American Huang Society of Reproductive Medicine, San Antonio, 2003.

34. Huang J Q, Krey L, **Licciardi F**. Single Blastocyst is an Effective Treatment Option. American Society of Reproductive Medicine, Philadelphia, 2004.

35. Flisser E, Kump L M, Krey L C, **Licciardi F**. Donor Age Does Not Impact the Success of Oocyte Donation Cycles. Fertil Steril 2004; 82:S211. Poster presentation at the American Society for Reproductive Medicine 60th Annual Meeting, 2004. Philadelphia, PA.

36. Fino E, Keegan D A, Noyes N, **Licciardi F**, Berkeley A S, Grifo J A
Reducing the Risk of Multiple Gestations with Ovulation Induction and Intrauterine Insemination: One Center's Experience. Annual meeting of the American Society of Reproductive Medicine, Montreal, 2005.

37. Lee H L, Adler A, Labella P, McCaffrey C, **Licciardi F**, Krey L C
The Effect of Media and Protein Supplements as Well as Day of Embryo Transfer (ET) on Monozygotic Twinning (MZT) Rates. Annual meeting of the American Society of Reproductive Medicine, Montreal, 2005.

38. Fino E, Noyes N, Keegan D A., Grifo J A, Berkeley A S, **Licciardi F**
Surgical Correction of Uterine Septum Improves Fertility and Pregnancy Outcome. Annual meeting of the American Society of Reproductive Medicine, Montreal, 2005.

39. Miles L M, Keitel M, Harris A, Jackson M, **Licciardi F.** (2005, August). Predictors of Distress in Women Undergoing Infertility Treatment. Poster presented at the 113th annual convention of the American Psychological Association, Washington D.C.

40. Flisser E, Levine BA, Krey LC and **Licciardi, F.**
Depot leuprolide acetate does not adversely affect oocyte donor stimulation
Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Page S409.

41. Knopman JM, Talebian S, Noyes N, Grifo JA, Krey LC and **Licciardi F**. Embryo and oocyte cryopreservation in female cancer patients: Does malignancy affect stimulation outcome? *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Pages S314-S315.*

Abstracts cont'd:

42. Keegan DA, Grifo JA, Lee T, **Licciardi F**, Naftolin F and Pevsner P. Direct matrix assisted laser desorption ionization (MALDI) identification of haptoglobin from culture

media of embryos that resulted in a live birth. *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Page S219.*

43. Salas J, Talebian S, Krey LC and **Licciardi F.** Gestational age at delivery is not affected by controlled ovarian hyperstimulation: A comparison of IVF and donor egg pregnancies *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Pages S186-S187.*

44. Flisser E, **Licciardi F** and Krey LC. The myth of the "clutch" donor *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Pages S183-S184.*

45. Grifo JA, Labella P, **Licciardi F**, Chang H, Lui H and Noyes N. Egg donors significantly under-report their weights. *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Page S138.*

46. Cho M and **Licciardi F.** Clinical results of an oocyte cryopreservation program *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Page S127.*

47. Conway DA, Flisser E, Krey LC and **Licciardi F.** Influence of endometrial thickness (ET) less than 7 millimeters on donor oocyte recipient outcomes. *Fertility and Sterility, Volume 86, Issue 3, Supplement 1, September 2006, Page S70.*

48. Mullin CM, Fino ME, Talebian S, Krey L, **Licciardi F** and Grifo JA. Age-related pregnancy outcomes in elective single embryo transfers (eSET) vs. double-embryo transfer (2ET) on day 5 in women <40 years of age. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Page S328.*

49. Kulp JL, Krey LC, Kwiatkowski A, **Licciardi F** and Noyes N. Human papilloma virus (HPV) and abnormal Pap smears at an infertility clinic: prevalence and association with fertility treatment outcome. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Pages S267-S268.*

50. Knopman JM, Talebian S, Krey LC, Berkeley AS, Grifo JA and **Licciardi F.** Do patients with successful donor embryo cycles have children from their supernumery cryopreserved embryos? *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Page S258.*

51. Gowda M, Talebian S, Noyes N, Berkeley AS, Grifo JA and **Licciardi F.** The role of oocyte donation in expanding the natural family. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Page S257.*

52. Knopman JM, Talebian S, Noyes N, Krey LC, Grifo JA and **Licciardi F.** The fate of cryopreserved donor embryos. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Pages S256-S257.*

Abstracts cont'd:

53. Mullin CM, Talebian S, Keegan D, Fino ME, Grifo JA and **Licciardi F.** Comparison of pregnancy outcomes in anonymous shared vs. exclusive donor oocyte in vitro

fertilization (IVF) cycles. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Page S132.*

54. Miller MJ, **Licciardi F**, J. Grifo, A.S. Berkeley and Noyes N. History of spontaneous abortion increases the risk of oocyte donation pregnancy loss. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Page S111.*

55. Reh AE, Krey L, Talebian S, **Licciardi F** and N. Noyes Ovarian stimulation in 2007: the evolving role of GNRH analogues at a large, university-based fertility center. *Fertility and Sterility, Volume 88, Supplement 1, September 2007, Pages S97-S98.*

Lectures:

October 1991	Lecture: Indications for IVF. American College of Obstetricians, Washington, DC
May 2, 1993	Infertility and Ovarian Cancer: The Facts on Women's Health Services. New York University Medical Center
May 4, 1993	Ask the Experts. RESOLVE Symposium New York, NY
June 29,1993	Infertility and Ovarian Cancer Seminar New York University Medical Center
Sept 23, 1993	Seminar: Infertility and Oocyte Donation RESOLVE and National Council of Jewish Women New York, NY
Oct 15, 1993	Seminar: The Infertile Couple New York, NY
Nov 18,1993	Time-Warner Health Seminar: The Diagnosis and Management of Infertility. New York, NY
Dec 5, 1993	Seminar: Infertility and IVF. Hoboken, NJ

Lectures cont'd:

Feb 20, 1994	Infertility Surgery. Pre-Operative Assessment Department of Nursing

	New York University School of Medicine
Mar 22, 1994	Women's Health Issues Women's Health Month. Featured Speaker: Veterans Administration Hospital New York NY
Mar 23, 1994	Infertility: From the diagnosis to IVF. New York University School of Medicine
Apr 20, 1994	IVF and Micro Manipulation Grand Rounds: Department of Urology New York University School of Medicine
May 1, 1994	Understanding your Blood Tests and Ultrasound. RESOLVE Symposium New York, NY
May 4, 1994	Infertility IVF And Oocyte Donation Department of Nursing New York University School of Medicine
Sept 14, 1994	New Techniques in the Management of Infertility Grand Rounds: Dept. of OB/GYN St. Vincent's Hospital & Medical Center New York, NY
Sept 21, 1994	IVF and Embryo Transfer Grand Rounds: Dept. of OB/GYN Brookdale Medical Center Brooklyn, NY
Sept 21, 1994	Infertility and its Therapy: Guest Lecturer: Metropolitan Museum of Art New York, NY
Dec 9, 1994	Infertility: The Basics through IVF Grand Rounds: New York Downtown Hospital. New York, NY
May 7, 1995	Ask the Experts. RESOLVE Symposium New York, NY

Lectures cont'd:

May 9, 1995	The Modern Management of Infertility. Grand Rounds: Dept. of OB/GYN St. Luke's /Roosevelt Hospital

	New York, NY
May 31, 1995	Endometriosis: An Update Grand Rounds: New York University Medical Center New York, NY
June 2 1995	New Perspectives in Infertility Grand Rounds: Dept. of OB/GYN Flushing Hospital, New York
Aug 10, 1995	The New Infertility Management Grand Rounds: Dept. of OB/GYN The Brooklyn Hospital & Medical Center Brooklyn, NY
Sept 21, 1995	Complications of Endometriosis Grand Rounds: Dept. of OB/GYN The Brooklyn Hospital & Medical Center Brooklyn, NY
June 27, 1996	ICSI and Micromanipulation. RESOLVE Seminar New York, NY
Sept 23, 1996	Oocyte Donation in the United States Italian Society of OB/GYN Bologna, Italy
Oct 18, 1996	New Perspectives in Endometriosis. Junior Fellow Meeting: American College of OB/GYN
Oct 1997	Medical Aspects of Oocyte Donation. RESOLVE Seminar New York, NY
Mar 1998	Is Oocyte Donation for me? RESOLVE Seminar New York, NY
Feb 1998	Understanding the CDC/SART data. RESOLVE Seminar New York, NY

Lectures cont'd:

Feb 1999	Understanding your IVF cycle RESOLVE Seminar New York, NY

Feb 1999	Heart Disease in women Women's O.W.N. Seminar New York, NY
Mar 1999	Oocyte Donation St. Barnabas Medical Conference Aruba, NA
Sept 1999	Oocyte Donation Update Grand Rounds NYU Medical Center New York, NY
Oct 1999	The Donor Registry Department of OB/GYN University of Alabama
Jan 2000	Fertility over 40 American Infertility Association New York, NY
May 2000	The Genetic Screening of Oocyte Donors The Art of Oocyte Donation Charleston, SC
Nov 2000	Advanced Infertility Therapy Resolve Valley Hospital, NJ
Nov 2000	Oocyte Donation Women's Own Seminar New York, NY
June 2002	Reproductive Options for Single Women NYU Medical Center New York, NY
April 2003	Oocyte Donation Grand Rounds Yale – New Haven Medical Center New Haven, Conn

Lectures cont'd:

May 2003	Egg Donor Rejection Grand Rounds NYU Medical Center New York, NY

May 2005	Modern Fertility Therapy Grand Rounds NYU Medical Center New York, NY
Nov 2005	The NYU Egg Donor System AIA

EXHIBIT N

CURRICULUM VITAE - GARRY R. CUTTING

I. Personal data

Full name:	Garry Richard Cutting, M.D.
Current appointments:	Professor of Pediatrics and Medicine Aetna/U.S. Healthcare Professor of Medical Genetics Director, Postdoctoral Training Programs in Medical Genetics Director, DNA Diagnostic Laboratory Johns Hopkins University School of Medicine Director, Translational Technology Core, Institute for Clinical and Translational Research
Birthdate and Place:	6/2/57; London, England
Citizenship:	Naturalized U.S. citizen 1979
Marital status:	Married 9/8/84; Maureen Cutting, Physician Assistant
Children:	Andrew and Allison, 8/11/87; Elizabeth, 7/21/92

II. Education and Professional Training

1975-1979	University of Connecticut Storrs, CT	B.S., Biology Magna Cum Laude
1979-1983	University of Connecticut Medical School Farmington, CT	M.D.
1983-1986	Johns Hopkins Hospital Baltimore, MD	Internship/Residency Pediatrics
1986-1989	Johns Hopkins University School of Medicine Baltimore, MD	Fellowship Pediatric Genetics

III. Professional Experience

1989-1993	Assistant Professor of Pediatrics Johns Hopkins University School of Medicine, Baltimore, MD
1990-1993	Assistant Professor of Medicine Johns Hopkins University School of Medicine, Baltimore, MD

1992-present	Director, Post-doctoral Training Programs in Medical Genetics Johns Hopkins University School of Medicine, Baltimore, MD
1993-1998	Associate Professor of Pediatrics and Medicine Johns Hopkins University School of Medicine, Baltimore, MD
1994-present	Director, DNA Diagnostic Laboratory Johns Hopkins University School of Medicine, Baltimore, MD
1998-present	Professor of Pediatrics and Medicine Johns Hopkins University School of Medicine, Baltimore, MD
1998-2008	Director, Genetic Residency Programs Johns Hopkins University School of Medicine, Baltimore, MD
1999-2003	Director, Cystic Fibrosis Genotyping Center Johns Hopkins University School of Medicine, Baltimore, MD
1999-2000	Interim Director, McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University School of Medicine, Baltimore, MD
2007-present	Director, Translational Technology Core, Institute for Clinical and Translational Research

IV. Licensure and Certification

1984	National Board of Medical Examiners
1985	Maryland Board of Medical Examiners
1987	American Board of Pediatrics
1990	American Board of Medical Genetics (Clinical Genetics, Biochemical Genetics and Molecular Genetics)

V. Honors and Awards

1974	Phi Beta Kappa and Phi Kappa Phi Honor Societies
1982	Lange Book Award for Scholarship, Medical School Third Year Clinical Rotations
1983	Linda Ives Award, Basic Science Research in Pediatrics
1990	Merck Clinician Scientist Award
1992	Election to the Society for Pediatric Research

1995	Election to the American Society for Clinical Investigation

VI. Professional Affiliations

1989	American Society of Human Genetics
1992	Association for Research in Vision and Ophthalmology
1993	American College of Medical Genetics, Founding Member

VII. National Committees

1993-present	Member, Program Committee; U.S. Cystic Fibrosis Foundation
1995-1998	Member, Program Committee; American Society of Human Genetics
1996-1997	Chairperson, Program Committee; American Society of Human Genetics
1996	Co-chairperson, Consensus Conference: Defining the Criteria for a CF Diagnosis, U.S. Cystic Fibrosis Foundation
1996-2000	Member, Mammalian Genetics Study Section, National Institutes of Health
1997	Chairperson, 47th Annual Meeting of the American Society of Human Genetics, Baltimore, MD
1998	Co-Chairperson, Genetic Testing for Cystic Fibrosis Committee, American College of Medical Genetics
1998	Member, Cystic Fibrosis Clinical Practice Working Group, American College of Obstetricians and Gynecologists
1999-2001	American Society of Human Genetics Representative to the Federation of American Societies for Experimental Biology Funding Conference
2001-2006	American Society of Human Genetics Representative to the Executive Committee of the Federation of American Societies for Experimental Biology

Miscellaneous Committees

2006	Leadership Development Program, JHU

VIII. Editorial Appointments

2007-present	Human Mutation, Co-Editor
2000-present	Current Molecular Medicine, Editorial Board
1998-present	Human Mutation, Electronic Editor
1997-present	Genetic Testing, Editorial Board
1991-present	Human Mutation, Communicating Editor

IX. Selected Invited Lectures and Symposia

1990-Present	Cystic Fibrosis Foundation Annual Williamsburg Conference, Williamsburg, VA Workshops: "CFTR and Electrolyte Physiology and Gene Therapy"
1991-Present	Annual Short Course in Medical and Experimental Mammalian Genetics, Jackson Laboratory, Bar Harbor, ME. Lectures: "Cystic Fibrosis" and "Genetics of Neuroreceptors and Ion Channels"
04/09/90 - 04/11/90	Istituto Giannina Gaslini, Sestri Levante, Italy. Presentation: "Identification of the CF gene: recent progress and new research strategies"
02/14/91	American Association for the Advancement of Science , Washington, DC. Symposium: "New Molecular Insights into 'Old' Genetic Disorders: Cystic Fibrosis"
09/19/91- 09/23/91	Royal College of Physicians and Surgeons of Canada, Quebec, Canada Symposium: "New Perspectives on Screening for Genetic Disease"
03/19/93	Georgetown University, Washington, DC FIDIA Georgetown Institute for the Neurosciences Presentation: "Molecular Biology of Novel GABA Receptor Subunits Preferentially Expressed In Retina"

04/07/94-04/08/94 Baylor College of Medicine, Houston, TX - Visiting Professor
Presentation: "The Association Between Mutation and Disease Severity in Cystic Fibrosis"

04/20/94-04/21/94 University of Florida, Gainesville, FL - Visiting Professor
Presentation: "Characterization of a Novel Class of GABA Receptor Subunits Expressed in Retina"

04/14/94-04/15/94 National Institute of Diabetes and Digestive and Kidney Disease, Bethesda, MD; Workshop on "Novel ATPases and Disease - ATP-Dependent Transporters and Emerging Superfamily. Presentation: "Mutations that Cause CF"

08/03/94-08/06/94 IV Latin American Cystic Fibrosis Congress Meeting, San Jose, Costa Rica. Presentations: "Gene therapy for CF update", "Epidemiology of Cystic Fibrosis in North America" and "DNA analysis"

09/08/94-09/10/94 Society of General Physiologists 48th Annual Symposium, Woods Hole, MA. Workshop on "Ion Channels and Genetic Diseases"
Symposium Presentation: "Correlating Ion Channels with Disease Using Genetic Linkage Analysis"

10/18/94-10/22/94 American Society of Human Genetics Annual Meeting, Montreal, Canada. Symposium Presentation: "Genotype/Phenotype Relationships in Cystic Fibrosis"

04/14/95 Institute of Child Health, "Aghia Sophia" Children's Hospital, Athens Greece.
Presentation: "Effect of CF Mutations upon CFTR Function"

04/19/95 Charles University Prague, Prague, Czech Republic.
Presentation: "Adeno-Associated Viral Vectors for Gene Therapy"

05/14/95-05/17/95 Association for Research in Vision & Ophthalmology Annual Meeting, Ft. Lauderdale, FL. Symposium Presentation: "Identification of Sequences Conferring Unique Properties to $GABA_C$ Receptors Formed of ρ Subunits"

01/19/96-01/21/96 American Academy of Allergy Asthma & Immunology, Scottsdale, AZ.
Presentation: "Genetic Aspects of Chronic Sinusitis"

06/16/96-06/21/96 XIIth International Cystic Fibrosis Congress, Jerusalem, Israel.
Symposium Presentation: "Mutation Analysis in Cystic Fibrosis"

08/17/96-08/22/96 9th International Congress of Human Genetics, Rio de Janeiro, Brazil.
Symposium Presentations: "Implications of the Multiple Functions of CFTR and Therapy for CF and Gene Therapy for Cystic Fibrosis"

11/03/96- 11/07/96	Conferences Philippe Laudat, Lyon, France. Symposium Presentation: "Consequences of CF Mutation Upon CFTR Functions"
04/14/97	NIH Consensus Conference: Genetic Testing for Cystic Fibrosis, Bethesda, MD. Presentation: "Ethnic distribution and phenotypic consequences of CF mutations"
05/09/97	First Annual Conference in Association with Vision Research: Molecular, Cellular and Genetic Approaches to Function and Dysfunction of the Retina, Fort Lauderdale, Fla. Symposium Presentation: Molecular composition of $GABA_C$ receptors
06/05/97	Cottle International Rhinology Centennial Conference, Philadelphia, PA. Presentations: "Molecular biology and rhinosinusitis" and "New directions in gene therapy for rhinology"
02/22/98	Annual Meeting, Biophysical Society, Kansas City, MO. Symposium Presentation: "Functional analysis of ClC-2 chloride channels stably expressed in cystic fibrosis airway cells"
5/23/98- 5/25/98	Departamento de Genetica Humana, Instituto Nacional de Saude, Lisbon, Portugal. Conference presentation: "Phenotypes Associated with Dysfunction of the Cystic Fibrosis Transmembrane Conductance Regulator"
7/10/98- 7/11/98	NHLBI Workshop, Bethesda, MD Workshop on Genetic Basis of Variability in the Progression and Outcome of Heart, Lung, and Blood Diseases. Presentation: Cystic Fibrosis
3/19/99- 3/20/99	2nd International Symposium on Inherited Diseases of the Pancreas, Cincinnati, Ohio. Presentation: "Genotype-Phenotype Relationships in Cystic Fibrosis"
9/12/99- 9/14/99	NIH Workshop, Bethesda, MD Workshop on ABC Transporters and Human Diseases. Presentation: "Cystic Fibrosis and Mutations in CFTR"
10/25/99 10/27/99	IPOKRaTES Course, Salzburg, Austria Course Title: Cystic Fibrosis - Recent Advances in Pediatric and Adult Issues.
2/19/00- 2/24/00	4th International Congress on Pediatric Pulmonology, Nice France. Conference presentation: "Novel Therapies to Circumvent the CFTR Defect"
3/20/00-	Mayo Clinic, Scottsdale, AZ. Seminar presentation: Signals for Apical

3/24/00	Localization in the C-terminus of the Cystic Fibrosis Transmembrane Conductance Regulator
02/22/01-02/23/01	Genetics Conference: New Diagnostic Challenges, Columbus, OH. Presentation: Inherited Disease Due to Mutations in Ion Channels
03/30/01-04/01/01	2nd Consensus Meeting CFTR Validation and Function Assays, Estoril, Portugal.
04/28/01-05/01/01	Pediatric Academic Society, Baltimore, MD. Presentation: Genetic Heterogeneity in CF
06/06/01-06/09/01	24th European Cystic Fibrosis Conference, Vienna, Austria Conference Presentation: "The CFTR Gene".
08/05/01-08/10/01	Gordon Research Conference, Newport, R.I. Presentation: Modifiers of Mendelian Disease-Cystic Fibrosis.
10/01/01-10/03/01	Yale University School of Medicine, New Haven, CT. Seminar Presentation: Identification of Apical Membrane Localization Signals in C-terminus of the Protein Responsible for Cystic Fibrosis.
04/18/02-04/19/02	Grand Rounds – Columbia University, New York, NY Presentation: "CFTR and It's Diseases".
05/30/02-05/31/02	NY Statewide Genetic Conference, Buffalo, NY Presentation: "Atypical Mutations in Cystic Fibrosis".
9/17/02-9/22/02	Manchester Paediatric Club, Manchester, UK Presentation: "What constitutes a cystic fibrosis diagnosis".
12/12/02-12/13/02	Nancy N. Hung MD Guest Professor in Cystic Fibrosis and Pediatric Pulmonology – St. Christopher's Hospital, Philadelphia, PA Presentation: "What is CF? The many faces of a diagnosis".
04/07/03-04/09/03	Foundation for Blood Research CF Course, Portland, ME Presentation: "The Essentials of Prenatal/Preconceptional Screening for Cystic Fibrosis".
5/8/03	Fifteenth Dr. Susan Lynn Shackman Memorial Lecturer, Johns Hopkins Department of Gynecology and Obstetrics Presentation: "Diagnosis for CF".
5/18/03	American Thoracic Society International Conference, Seattle, WA Presentation: "Cystic Fibrosis without CFTR mutations".

5/29/03	NJ Department of Health and Senior Services, New Brunswick, NJ Monmouth Medical Center and St. Barnabas Medical Center Presentation: "The Genetics of Cystic Fibrosis".
1/14/04- 1/25/04	Grand Rounds – The University of Oklahoma, Oklahoma City, OK Presentation: Causes of Phenotype Variation in a "Monogenic" Disorder.
4/30/04- 5/3/04	2004 European Cystic Fibrosis Society Conference, Tomar, Portugal Presentation: "CFTR and It's Diseases"
6/4/04- 6/8/04	Cystic Fibrosis Foundation – Williamsburg, Conference, Williamsburg, VA Presentation: ENaC Activity and CF
6/13/04 - 6/17/04	27th European Cystic Fibrosis Conference, Birmingham, UK Presentation: "Phenotype: Genes or Environment?"
7/15/04- 7/16/04	Children's Hospital and Regional Medical Ctr. CF Conference, Seattle, WA Presentation: "CFTR mutation analysis", and "The contribution of genes and environment to CF phenotype variability".
7/16/04- 7/18/04	Roche Molecular Seminar, Oakland, CA Presentation
9/13/04- 9/14/04	Massachusetts General Hospital Symposium on CF, Harvard Club, Boston, MA Presentation: "New insights into the causes of variation of the CF phenotype".
10/14/04- 10/16/04	2004 North American CF Conference, St. Louis, MO Presentation: "What have we learned from Correlating Genotype to Phenotype?" and "CF Modifiers: Comparing Variation Between Siblings"
2/9/05- 2/10/05	Solvay Pharma, Montreal, Canada Presentation: "Contributions of Genes and Environment to Variation in CF".
3/16/05	Grand Rounds – The Georgetown University Hospital, Washington, DC Presentation: "Development and Validation of Preimplantation Genetic Diagnosis for Clinical Applications"
5/17/05- 5/18/05	Seminar – Dartmouth Medical School, Hanover, NH Presentation #1: "Contribution of Genes and Environment to Variations In Cystic Fibrosis" Presentation #2 to COBRA audience: "Elucidating the Mechanism Underlying Localization of the CFTR C-Terminus to Apical Membranes"

6/3/05- 6/7/05	Cystic Fibrosis Foundation – Williamsburg Conference, Williamsburg, VA Presentation: "Genetic and Environmental Modifiers"
7/29/05	46th Annual Short Course, Jackson Lab – Bar Harbor, ME Presentation: "DNA and Diagnostic Screening"
10/20/05 - 10/23/05	19th NACF Conference – Baltimore, MD Presentation: "Genotypes/Phenotypes".
11/11/05	Principles of Developmental Biology – Johns Hopkins University, MD Presentation: "Clinical Correlation: Prenatal Genetic Diagnosis"
3/23/06 – 3/26/06	2006 ACMG Clinical Genetics Meeting – San Diego, CA Presentation #1: "The New Genetics: Practicing Medical Genetics in the Era of Genomics". Presentation #2: "the contribution of Genetic Variation in Rare Disease Genes to common diseases: Chronic Sinusitis, Lung disease, Infertility, and CFTR".
4/28/06	Department of Pathology Seminar – Johns Hopkins University, MD Presentation: "Overview of Genetic Testing".
6/2/06 - 6/6/06	Williamsburg Conference – Williamsburg, VA Presentation: "From the Home, to the Bench, to the Clinic"
7/16/06 - 7/28/06	47th Annual Short Course, Jackson Lab – Bar Harbor, ME Presentation: "DNA and Diagnostic Screening"
11/12/06	Emory – Genetic Seminar Series – Atlanta, GA Presentation: "Genetic & Non-Genetic Modifiers of Disease Severity in CF".
3/20/07- 3/28/07	Consensus Conference on Molecular Analysis in CF – Garda Lake, Italy Presentation: "CF Mutation Analysis in the Clinical Setting"
4/12/07- 4/14/07	12th Annual Meeting of the Council of Physicians/Scientists – Dallas, TX Presentation #1: "Principals & Practice of Pre-implantation Genetic Diagnosis for CF" Presentation #2: "Aspects of Setting up a PDG Laboratory"
5/7/07- 5/8/07	CF Foundation Conference – Diagnosis Criteria for CF – Bethesda, MD Presentation: "Issues surrounding DNA Analysis and Interpretation"
6/1/07- 6/5/07	Williamsburg Conference – Williamsburg, VA Presentation: Genotype/Phenotype Relationships in CF: Future Lessions"
7/7/07 - 7/27/07	48th Annual Short Course, Jackson Lab – Bar Harbor, ME Presentation: "DNA and Diagnostic Screening"

8/11/07- 8/14/07	7th Australian CF Conference – Sydney, Australia Presentation #1: "What is new in CF Genetic Research" Presentation #2: "From Genes to Treatment" Presentation #3: "Gene Transfer to People with CF"
10/3/07- 10/6/07	21st Annual NACF Conference – Anaheim, CA Presentation: "Is it all Genetics?"
1/16/08	Newborn Screening: Issues and Answers Series - Bethesda, MD Presentation: Reactor Discussion – Reactor 2- "False-Negative/False Positive Screening Reports" – "Appropriate Mutation Panel for DNA Testing in Screening Protocol"
4/9/08- 4/13/08	European CF Society Conference (ECFS) – Douro, Portugal Presentation: "The US Initiative to Functionally Characterize all CFTR Mutations in the Same Cellular System"
5/25/08- 5/29/08	Human Variome Project – Costa Brava, Spain Presentation: "Complementing Mutation-Driven Databases with Phenotype-Rich Repositories: The Clinical and Functional TRanslation of CFTR (CFTR2) Project"
5/30/08- 6/3/08	Williamsburg Conference – Williamsburg, VA Presentation: "Expression and Analysis of Full Length CFTR"
6/11/08- 6/14/08	31st European CF Conference – Prague, Czech Republic Presentation: "Diabetes in CF"
7/20/08 - 8/1/08	49th Annual Short Course, Jackson Lab – Bar Harbor, ME Presentation: "Genetic Modifiers of CF"
10/22/08- 10/24/08	1st Advances in Rare Bone Diseases Scientific Conference – NIH-Bethesda, MD Presentation: "Modifier Genes: Lessons from Cystic Fibrosis"
10/23/08- 10/25/08	22nd Annual NACF Conference – Orlando, FL Presentation: "Finding Genetic Modifiers of CF Lung Disease Using Genome-wide Linkage"
10/28/08- 10/30/08	2nd Al Ain International Genetics Conference – Dubai, United Arab Emirates Presentation #1: "Pre-implantation genetic diagnosis (PDG): Basics and New Technologies" Presentation #2: "Interactions Among Genetic Modifiers and Environment Underlying Variation in the Severity of a Single Gene Disorder: Cystic Fibrosis"
4/15/09- 4/19/09	New Frontiers in Basic Science of CF – Tavira, Protugal Presentation: "Genes and the Environment"

5/15/09- 5/20/09	American Thoracic Society 2009 Conference – San Diego, CA Presentation: "Uses and Misuses of Genetic Testing in CF Diagnosis"
6/10/09- 6/13/09	32nd European CF Conference – Brest, France Presentation: "CFTR2 2009 Genotype/Phenotype Correlations for the Clinician"
7/24/09- 7/31/09	50th Annual Short Course, Jackson Lab – Bar Harbor, ME Presentation: "Genetic Modifiers of CF"
8/29/09- 9/2/09	International Congress of Inborn Errors of Metabolism 2009 – San Diego, CA Presentation: "Cystic Fibrosis as a Model for Complex Diseases"

Scientific Reviews:

Journals:

American Journal of Human Genetics
American Journal of Medical Genetics
Biotechniques
Genomics
Human Genetics
Human Molecular Genetics
Journal of Clinical Investigation
Nature
Nature Genetics
New Engl. Journal of Medicine
Nucleic Acids Research
Pediatrics Human Mutation
Prenatal Diagnosis
Proceedings of the National Academy of Sciences, USA

Grants:

National Institutes of Health:
Human Genome Center, ELSI Study Section, 1992
Parent Review Committee for SCOR Program, NHLBI, 1993
Mammalian Genetics Study Section: Ad Hoc, 1993, 1995
Regular member 1996-2000

Cystic Fibrosis Foundation:
University of Michigan RDP Site Visit, 1991
Ad Hoc Reviewer, 1992 - 1994, 1995, 1998-2001

X. Research Support

National Institutes of Health, R37 DK44003, MERIT Award: The molecular genetics of Cystic Fibrosis, 2003-2013. Principal Investigator.

National Institute of Health, R01 HL68927. Genetic Modifiers of Cystic Fibrosis: Sibling Study 9/30/01 – 3/31/12. Principal Investigator.

National Institutes of Health, T32 GM07471, Medical Genetics Fellowship Training Grant,

1977-2011. Principal Investigator.

National Institutes of Health, UL1 RR025005, Clinical Translational Science Award. Project: Technology Translation Core 9/30/07-9/20/12. Principal Investigator

Flight Attendants Medical Research Institute, 062553_CIA Clinical Innovator Award. 7/1/07 – 6/30/10 Principal Investigator

Cystic Fibrosis Foundation, CUTTIN08A0, CFTR2 Project 2008-2010

Past:

Merck/Johns Hopkins University School of Medicine Clinician Scientist Award. Molecular genetics of cystic fibrosis, 1990-1992. Principal Investigator

National Institutes of Health, R01 DK39635, The molecular genetics of cystic fibrosis, 1987-90. Co-Principal Investigator

National Institutes of Health R01 EY09531, Molecular biology of retina-specific GABA receptors, 1992-2000. Principal Investigator.

National Institutes of Heath, P50 DK489771994-1999. Principal Investigator Project I: Investigating alternate chloride channels in CF.

National Institutes of Health, P01 AI37163, Project I : Mutations in the CFTR gene in patients with chronic sinusitis; 1995-2003, Principal Investigator.

National Institute of Health, U01 HL66615. Project 1: Applied Genomics in Cardiopulmonary Disease; 2000-2004, Co-Investigator.

Cystic Fibrosis Foundation, R0209C-1, The cloning of the chloride channel defective in cystic fibrosis, 1989-91. Principal Investigator

Cystic Fibrosis Foundation, R0251-1, Molecular studies of chloride channels, 1991-1994. Principal Investigator

Cystic Fibrosis Foundation, Integration of Recombinant AAV-CFTR Vectors, 1995-1996. Principal Investigator

Cystic Fibrosis Foundation. CF Genotyping Center, 1998-2003. Principal Investigator – no salary support.

Cystic Fibrosis Foundation. Genetic Modifiers of Cystic Fibrosis: Twin Study, 2000-2002. Principal Investigator.

Cystic Fibrosis Foundation. CUTTIN06P0. Genetic Modifiers of CF: Twin/Sibling Study. 9/1/06 –8/31/08. Principal Investigator.

XI. Teaching Experience

Formal Courses:
Graduate Student Seminar, Johns Hopkins University: "Molecular Mechanisms of Disease"
CoDirector 1991 - 1994
Director 1995 - 2000

Graduate Student Course, Johns Hopkins University: "Advanced Topics in Human Genetics"
Lecturer 1993 - present

Medical School Course, Johns Hopkins University: "Molecules and Cells"
Lecturer 1994 - present

Research Trainees:

PAST:

Name	Position	Years	Degree	Research Project	Present Position
Hamosh A	Postdoc	89-92	*MD,MPH*	Genotype/phenotype studies in CF	Professor, Pediatrics Johns Hopkins School of Med.
McIntosh I	Postdoc	91-94	*PhD*	Molecular genetics of CF	Professor of Genetics, Univ. of the Caribbean
Friederick C	Postdoc	92-94	*MD,PhD*	Mapping of the human GABA rho1 gene	Assistant Professor, Medicine University of Pennsylvania
Macek M Jr	Postdoc	92-95	*MD,PhD*	Mutation identification in CF patients	Head of National Center for CF, Prague, Czech Republic
Cid Soto P	Postdoc	93-95	*MD*	Molecular biology of a voltage-gated chloride channel	Assistant Professor, Medicine University of Chile
Miller P	Postdoc	93-95	*MD*	Mutation analysis of pulmonary diseases	Associate Professor, Medicine UCLA/West LA, VA Medical Ctr.
Enz R	Postdoc	96-98	*PhD*	Molecular biology of GABA rho receptors	Assistant Professor, Univ. Erlangen-Nuernberg, Germany
Hackam A	Predoc (Hum.Gen)	93-97	*PhD awarded*	Assembly of GABA rho subunits	Assistant Professor Bascom-Palmer Inst., U. Miami, FL
Fulmer S	Predoc (Hum.Gen)	93-97	*PhD awarded*	Consequences of mutations upon CFTR function	Dept. Of Genetics, Stanford Univ., CA
Ross B	Postdoc	96-99	*MD*	In utero gene therapy	Medical Staff, Beverly Hosp., Montebello, CA
Mickel J	Postdoc	95-00	*PhD*	Localization signals in CFTR	Assist. Professor, Pediatrics, Johns Hopkins School of Medicine
Landrum M	Predoc (Hum.Gen)	95-00	*PhD Awarded*	Integration of recombinant AAV vectors	Staff, National Center for Biotechnology, NIH, Bethesda, MD
Wu Y	Postdoc	97-01	*MD,Ph.D.*	Transcriptional regulation of GABA rho genes in retina	Research Scientist, Biopharma, NJ
Milewski M	Postdoc	97-01	*PhD*	Interactions between CFTR and other Cl- channels	Research Scientist, Univ of Warsaw, Poland
Wang X	Postdoc	97-01	*MD,PhD*	Molecular genetics of sinusitis	Director, DNA Diagnostic Lab, Univ. of Minn.
Yaghmai R	Postdoc	98-00	*MD,PhD*	In vitro generation of DNA binding protein libraries	Asst. Prof., Univ. Calif., San Diego
Hoover-Fong J	Postdoc	01-02	*MD*	Fatty Acid Abnormalities in CF	Assistant Professor, Pediatrics, Johns Hopkins School of Medicine
Buranawuti K	Postdoc	01-03	*MD*	Molecular Genetics	Postdoc in DNA Lab
Hefferon T	Predoc (Hum.Gen)	99-03	*PhD Awarded*	CFTR Splicing, Exon 9	Postdoc, NIH
Groman J	Predoc (Hum.Gen)	00-03		Modifier genes for cystic fibrosis	Consultant, Private Industry
McWilliamsR	Predoc (Epidem.)	00-04		Cystic Fibrosis Twin/Sib Study	Assistant Professor, School of Public Health, Rutgers
Larusch J	Predoc (Hum.Gen)	00-08	*PhD Awarded*	C-Terminal associated proteins Identification of CFTR interacting proteins that mediate localization	Post-doc Univ of Pittsburgh
Hsu S	Postdoc	02-07	*MD,PhD*	G Protein dysfunction and CF	Pediatric Endocrinology, Private Practice,
Sheridan M	Predoc (CMM)	03-08	*PhD Awarded*	Non-classic CF	Post-doc Univ. of Pennsylvannia

Krasnov K	Predoc (CMM)	03-08	*PhD Awarded*	CFTR Protein Localization	Technical Advisor, Venable LLP
McDougal K	Predoc (Hum Gen)	03-08	*PhD Awarded*	Modifiers of CF	Postdoc, JHU, School of Public Health
Vanscoy L.	Postdoc	05-07	*MD*	CF Twin/Sibling Study	US Navy
PRESENT:					
Bremer, L.	Predoc	05-		Modifier Genes of CF	Predoc Student in the lab
Blackman S.	Postdoc	05-	MD/PhD	Modifier Genes of CF	Assitant Professor, Dept of Pediatrics, Johns Hopkins
Collaco M.	Postdoc	05-	MD	CF Twin/Sibling Study	Postdoc Fellow in the lab
Sosnay P.	Postdoc	05-	MD	CF Twin/Sibling Study	Assistant Prof., Dept of Pediatrics, Johns Hopkins
Greene D.	Postdoc	05-	MD	CFTR Trafficking	Assistant Prof., Dept of Medicine, Johns Hopkins
Doshi, V	Postdoc	07-	MD	Infection Phenotype in CF	Postdoc Fellow in the lab
Sharma N.	Postdoc	08-	MD	CFTR Trafficking	Postdoc Fellow in the lab
Kottoor, V.	Postdoc	09-	MD	Modifiers of CF	Postdoc Fellow in the lab

DNA LABORATORY TRAINEES:

Name	Position	Years	Degree	Present Position
Doheny K	Postdoc	95-96	PhD	Director of Technology & Development Center for Inherited Disease Research at Bayview, Johns Hopkins University/Natl. Inst. Of Health
Goodman B	Postdoc	95-96	PhD	Instructor, Obstetrics/Gynecology, Johns Hopkins
Chong S	Postdoc	96-96	PhD	Assistant Professor, National University, Singapore
Schmeckpeper B	Postdoc	96 – 98	PhD	Assistant Professor, Medicine, Johns Hopkins
Cordero D	Postdoc	97 - 98	MD	Assistant Professor, Columbia University, NY
Boyadjiev S	Postdoc	98-99	MD	Assistant Prof. Johns Hopkins University, School of Medicine
Yaghmai R	Postdoc	98-99	MD,PhD	Resident, Medicine, Johns Hopkins Bayview
Bober M	Postdoc	99-00	MD,PhD	Assoc. Prof. Univ. TX, Southwestern Med. Ctr.
Wang XJ	Postdoc	00-01	MD,PhD	Instructor, Pediatrics Johns Hopkins School of Medicine
Steinberg S	Postdoc	02-04	MD	Assistant Professor, Kennedy Krieger Institute, Baltimore, MD
Buranawuti K	Postdoc	03-06	MD	Postdoc in DNA Laboratory
Macaya D	Postdoc	03-05	PhD	Postdoc in DNA Laboratory
Al-Saif A	Postdoc	04-06	MD	Postdoc in DNA Laboratory

XII. Publications

1. Cutting GR, Antonarakis SE, Youssoufian H and Kazazian H Jr. The accuracy and limitations of Pulsed Field Gel Electrophoresis in sizing partial deletions of the factor VIII gene. **Molecular Biology and Medicine** (1988), 5:173-184.

2. Cutting GR, Kazazian H Jr, Antonarakis SE, Killen PD, Yamada Y and Francomano CA. Macrorestriction mapping of human collagen genes COL4A1 and COL4A2 on chromosome 13q34. **Genomics** (1988), 3:256-263.

3. Jabs EW, Goble CA, Cutting GR. The macromolecular organization of human centromeric region reveals high frequency, polymorphic macro-DNA repeats. **Proc Natl Acad Sci (USA)** (1989), 86:202-206.

4. Cutting GR, Antonarakis SE, Beutow KH, Kasch LM, Rosenstein BJ, Kazazian H Jr. Analysis of DNA polymorphism haplotypes linked to the Cystic Fibrosis locus in North American Black and Caucasian families support the existence of multiple mutations of the Cystic Fibrosis gene. **Am J Hum Genet** (1989), 44:307-318.

5. Cutting GR, McGinniss MJ, Kasch LM, Tsipouras P, Antonarakis SE. Physical mapping by PFGE localizes the COL3A1 and COL5A2 genes to a 35kb region on chromosome 2. **Genomics** (1990), 8:407-410.

6. Cutting GR, Kasch LM. Worldwide survey of the deltaF508 mutation-report from the Cystic Fibrosis Genetic Analysis Consortium. **Am J Hum Genet** (1990), 47:354-359.

7. Cutting GR, Kasch LM, Rosenstein BJ, Tsui L-C, Kazazian H and Antonarakis SE. Two patients with Cystic Fibrosis, nonsense mutations in each Cystic Fibrosis gene, and mild pulmonary disease. **New Engl J Med** (1990), 323:1685-1689.

8. Cutting GR, Kasch LM, Rosenstein BJ, Zielensky J, Tsui L-C, Antonarakis SE, Kazazian H Jr. A cluster of Cystic Fibrosis mutations in the first nucleotide binding fold domain of the Cystic Fibrosis conductance regulator protein. **Nature** (1990), 346:366-369.

9. Beaudet AL, Perciaccante RG, Cutting GR. Homozygous nonsense mutation causing cystic fibrosis with uniparental disomy. **Am. J. Hum. Genet.** (1991) 48:1213.

10. Nunes V, Gaona MC, Mana P, Casals T, Cutting GR, Estivill X: Prenatal diagnosis of cystic fibrosis by simultaneous analysis of two different mutations. **Prenatal Diagnosis** (1991) 11:671-672.

11. McColley SA, Rosenstein BJ, Cutting GR. Differences in Expression of Cystic Fibrosis in Blacks and Whites. **Am J Dis Child** (1991), 145:94-97.

12. Zeitlin PL, Lu L, Hwang TC, Rhim J, Craig R, Cutting GR, Stetten G, Kieffer KA, Guggino WB. A Cystic Fibrosis bronchial epithelial cell line: Immortalization by Adeno-12 SV-40 infection. **Am J Resp Cell and Mol Biol** (1991), 4:313-319.

13. Reiss J, Cooper DN, Bal J, Slomski R, Cutting GR, Krawczak M. Discrimination between recurrent mutation and identity by descent: application to point mutations in exon 11 of the CFTR gene. **Human Genetics** (1991), 87:457-461.

14. Francomano CA, Cutting GR, McCormick MK, Chu ML, Timpl R, Hong HK, Antonarakis SE. The COL6A1 and COL6A2 genes exist as a gene cluster and detect highly informative DNA polymorphisms in the telomeric region of human chromosome 21q. **Human Genetics** (1991), 87:162-166.

15. Chu C-S, Trapnell BC, Murtagh JJ Jr, Moss J, Dalemans W, Jallat S, Mercenier A, Pavirani A, Lecocq J-P, Cutting G, Guggino WB and Crystal RG. Variable deletion of exon 9 coding sequences in Cystic Fibrosis gene mRNA transcripts in normal bronchial epithelium. **EMBO J.** (1991), 10:1355-1363.

16. Graham CA, Goon PKC, Hill AJM, Cutting GR, Curristin S, Nevin NC. Identification of a new mutation (R297Q) in exon 7 of the CFTR gene in a Northern Ireland family. **J Med Genet** (1991) 28:571.

17. Hamosh A, Trapnell BC, Zeitlin PL, Montrose-Rafizadeh C, Rosenstein BJ, Crystal RG, and Cutting GR. Severe deficiency of CFTR mRNA carrying nonsense mutations R553X and W1316X in respiratory epithelial cells of patients with cystic fibrosis. **J Clin Invest** (1991) 88:1880-1885.

18. Dietz HC, Cutting GR, Pyeritz RE, Maslen CL, Sakai LY, Corson GM, Puffenberger EG, Hamosh A, Nanthakumar EJ, Curristin SM, Stetten G, Meyers DA, Francomano CA. Marfan syndrome caused by a recurrent denovo missense mutation in the fibrillin gene. **Nature** (1991), 352:337-339.

19. Cutting GR, Lu L, O'Hara B, Kasch LM, Donovan D, Shimada S, Antonarakis SE, Guggino WB, Uhl GR, Kazazian H Jr. Cloning of the gamma-aminobutyric acid (GABA) rho_1 cDNA: A GABA receptor subunit highly expressed in the retina. **Proc Natl Acad Sci (USA)** (1991), 88:2673-2677.

20. Dietz HC, Pyeritz RE, Kendzior RJ, Puffenberger EG, Corson G, Sakai LY, Francomano CA, Cutting GR. Marfan phenotype variability in a family segregating a missense mutation in the EGF-like motif of the fibrillin gene. **J Clin Invest** (1992), 89:1674-1680.

21. Dietz HC, Saraiva J, Pyeritz RE, Cutting GR, Francomano CA. Clustering of fibrillin 15 missense mutations causing the Marfan syndrome at residues with significance for calcium binding in EGF-like domains. **Hum Mutation** (1992), 1:366-374.

22. Abeliovich D, Lavon IP, Lerer I, Cohen T, Springer C, Avital A, Cutting GR. Screening for five mutations detects 97% of CF chromosomes and predicts a carrier frequency of 1:29 in the Jewish Ashkenazi populations. **Am J Hum Genet** (1992) 51:951-956.

23. Chu C-S, Trapnell BC, Curristin SM, Cutting GR, Crystal RG. Extensive post-transcriptional deletion of the coding sequences for part of nucleotide-binding fold 1 in respiratory epithelial

mRNA transcripts of the cystic fibrosis transmembrane conductance regulator gene is not associated with the clinical manifestations of cystic fibrosis. **J Clin Invest** (1992) 90,785-790.

24. Cozens AL, Yezzi MJ, Yamaya M, Steiger D, Wagner JA, Garber SS, Chin L, Simon EM, Cutting GR, Gardner P, Friend DS, Basbaum CB, Gruenert DC. A transformed human epithelial cell line that retains tight junctions post crisis. **In Vitro Cell Dev Biol** (1992), 28A:735-744.

25. Cutting GR, Curristin SM, Nash E, Rosenstein BJ, Lerer I, Abeliovich D, Hill A, Graham C. Analysis of four diverse population groups indicates that a subset of cystic fibrosis mutations occur in common among Caucasians. **Am J Hum Genet** (1992), 50:1185-1194.

26. Cutting GR, Curristin S, Zoghbi H, O'Hara B, Seldin MF, Uhl GR. Identification of a putative gamma aminobutyric acid (GABA) receptor subunit rho_2 cDNA and colocalization of the genes encoding rho_2 (GABBR2) and rho_1 (GABRR1) to human chromosome 6q14-q21 and mouse chromosome 4. **Genomics** (1992), 12:801-806.

27. Hamosh A, King TM, Rosenstein BJ, Corey M, Levison H, Durie P, Lap-Chee T, McIntosh I, Keston M, Brock DJH, Macek M Jr, Zemkova D, Krasnicanova H, Vavrova V, Macek M Sr, Golder N, Schwarz MJ, Super M, Watson EK, Williams C, Bush A, O'Mahoney SM, Humphries P, DeArce MA, Reis A, Burger J, Stuhrmann M, Schmidtke J, Wulbrand U, Dork T, Tummler B, Cutting GR. Cystic fibrosis patients bearing both the common missense mutation gly→asp at codon 551 and the ΔF508 mutation are clinically indistinguishable from ΔF508 homozygotes, except for decreased risk of meconium ileus. **Am J Hum Genet** (1992) 51:245-250.

28. Hamosh A, Rosenstein BJ, Cutting GR. CFTR nonsense mutations G542X and W1282X associated with severe reduction of CFTR mRNA in respiratory epithelial cells. **Hum Mol Genet** (1992) 1:542-544.

29. Lerer I, Sagi M, Cutting GR, Abeliovich D. Cystic fibrosis mutations in Jewish patients: deltaF508 and G542X. **J Med Genet** (1992), 29:131-133.

30. Macek, M, Hamosh, A, Kiesewetter S, McIntosh, I, Rosenstein, BJ, Cutting GR. Identification of a novel nonsense mutation (L88X) in exon 3 of the CFTR gene on a native Korean cystic fibrosis chromosome. **Hum Mutation** (1992) 1:501-502

31. Montrose-Rafizadeh C, Blackmon DL, Hamosh A, Oliva MM, Hawkins AL, Curristin SM, Griffin CA, Yang VW, Guggino WB, Cutting GR, Montrose MH. Regulation of CFTR gene transcription and alternative RNA splicing in a model of developing intestinal epithelium. **J Biol Chem** (1992) 267:299-305.

32. Shimada S, Cutting GR, Uhl GR. GABA A or C receptor?: GABA rho_1 receptor RNA induces bicuculline, barbiturate and benzodiazepine-insensitive GABA responses in Xenopus oocytes. **Mol Pharm** (1992), 41:683-687.

33. Zeitlin PL, Crawford I, Lu L, Woel S, Cohen ME, Donowitz M, Montrose M, Hamosh A, Cutting GR, Gruenert D, Huganir R, Maloney P, Guggino WB. CFTR protein expression in primary and cultured epithelia. **Proc Natl Acad Sci (USA)** (1992), 89:344-347.

34. Chu CS, Trapnell BC, Curristin S, Cutting GR, Crystal RG. Genetic basis for variable exon 9 skipping in cystic fibrosis transmembrane conductance regulator mRNA. **Nature Genetics** (1993), 3:151-156.

35. Dietz HC, Francomano CA, Kendzior RJ Jr, Pyeritz, RE, Valle DV, Cutting GR. The skipping of constitutive exons in vivo induced by nonsense mutations. (1993) **Science** 259:680-683.

36. Kiesewetter S, Macek M Jr., Davis C, Curristin SM, Chu C-S, Graham C, Shrimpton AE, Cashman SM, Tsui L-C, Mickle J, Amos J, Highsmith WE, Shuber A, Witt DR, Crystal RG and Cutting GR. A mutation in the cystic fibrosis transmembrane conductance regulator gene produces different phenotypes depending on chromosomal background. **Nature Genetics** (1993), 5:274-278.

37. Kusama T, Wang T-L, Guggino WB, Cutting GR and Uhl GR. GABA ρ_2 receptor pharmacological profile: GABA recognition site similarities to ρ_1. **Europ J Pharm-Mol Pharm** (1993), 245:83-84.

38. O'Hara BR, Smith SS, Cutting GR, Uhl GR. Population differences in dopamine D2 receptor alleles suggest caution for associational studies. **Hum Hered** (1993), 43:209-218.

39. Genotype-Phenotype consortium (Hamosh A, Rosenstein BJ, Nash E, Curristin SM and Cutting GR). Correlation between genotype and phenotype in cystic fibrosis: Analysis of seven common mutations. **New Eng J Med** (1993), 329: 1308-1313.

40. Carroll TP, McIntosh I, Zeitlin PL, Cutting GR, Guggino WB. Transmembrane and nucleotide-binding fold mutations alter the channels characteristics of the cystic fibrosis transmembrane conductance regulator expressed in Xenopus oocytes. **Cell Physiol Biochem** (1994), 4:10-18.

41. Ramsey BW, Boat TF, Accurso F, Bennett W, Boucher R, Brody A, Crystal R, Cutting GR, Davis P, Dorkin H, Fleming T, Kaplan RM, Knowles M, Konstan MW, Laube B, McElvaney G, Morgan W, Moss R, Orenstein D, Pencharz P, Rosenstein B, Schidlow D, Smith A, Spino M, Stallings V, Stecenko A, Terrin M, Weatherly M, Wilmott R, Wilson C, and Wood R. Outcome Measures for Clinical Trials in Cystic Fibrosis. **J Ped** (1994), 124:177-192.

42. Wang T-L, Guggino WB, Cutting GR A novel γ-aminobutyric acid receptor subunit (rho_2) cloned from human retina forms bicuculline-insensitive homooligomeric receptors in Xenopus oocytes. **J Neurosci** (1994), 14:6524-6531.

43. Schwiebert EM, Flotte T, Cutting GR, Guggino WB. CFTR and outwardly rectifying chloride channels contribute to whole cell chloride currents in normal airway epithelial cells and are defectively regulated in cystic fibrosis. **Am J Physiol (Cell)** (1994), 266: 1464-1477.

44. Smit LS, Strong TV, Wilkinson DJ, Macek Jr M, Mansoura MK, Wood DL, Cole JL, Cutting GR, Cohn JA, Dawson DC, Collins FS. Missense mutation (G480C) in the CFTR gene associated with protein mislocalization but normal chloride channel activity. **Hum Molec Genet** (1995), 4(2): 269-273.

45. Cid LP, Montrose-Rafizadeh Ch, Smith DI and Cutting GR. Cloning of a putative human voltage-gated chloride channel (ClC-2) cDNA widely expressed in human tissues. **Hum Molec Genet** (1995), 4(3):407-413.

46. Carroll TP, Morales MM, Fulmer SB, Allen SS, Flotte TR, Cutting GR, Guggino WB. Alternate translation initiation codons can create functional forms of CFTR. **J Biol Chem** (1995), 270(20):11941-11946.

47. Schwiebert EM, Egan ME, Hwang T-H, Fulmer SB, Allen SS, Cutting GR, and Guggino WB. CFTR regulates outwardly rectifying chloride channels through an autocrine mechanism involving ATP. **Cell** (1995), 81(7):1063-1073.

48. Fulmer SB, Schwiebert EM, Morales MM, Guggino WB and Cutting GR. Two cystic fibrosis transmembrane conductance regulator mutations have different effects on both pulmonary phenotype and regulation of outwardly rectified chloride currents. **Proc Natl Acad Sci USA** (1995), 92:6832-6836.

49. Wang T-L, Hackam A, Guggino WB, Cutting GR. A single histidine residue is essential for zinc inhibition of GABA ρ1 receptors. **J Neurosci** (1995), 15:7684-7691.

50. Wang T-L, Hackam A, Guggino WB, Cutting GR. A single amino acid in γ-aminobutyric acid ρ1 receptors affects competitive and noncompetitive components of picrotoxin inhibition. **Proc Natl Acad Sci USA** (1995), 92:11751-11755.

51. Morales MM, Carroll TP, Morita T, Schwiebert EM, Wilson PD, Lopes AG, Stanton BA, Dietz HC, Cutting GR, Guggino WB. Both wild-type and a functional isoform of CFTR are expressed in kidney. **Am J Physiol** (Renal, Fluid and Electrolyte) (1996), 270(39):F1038-F1048.

52. Kearns,W.G.; Afione,S.A.; Fulmer,S.B.; Pang,M.C.; Erikson,D.; Egan,M.; Landrum,M.J.; Flotte,T.R.; Cutting,G.R. Recombinant Adeno-Associated Virus (AAV-CFTR) Vectors Do Not Integrate in a Site-Specific Fashion in an Immortalized Epithelial Cell Line. **Gene Therapy** (1996), 3:748-755.

53. Afione SA, Conrad CK, Kearns WG, Chunduru S, Adams R, Reynolds TC, Guggino WB, Cutting GR, Carter BJ and Flotte R. An *in vivo* model of adeno-associated virus vector persistence and rescue. **J Virol** (1996), 70:3235-3241.

54. Miller PW, Hamosh A, Macek Jr, M., Greenberger PA, MacLean J, Walden SM, Slavin RG and Cutting GR. Cystic fibrosis transmembrane conductance regulator (CFTR) gene mutations in allergic bronchopulmonary aspergillosis. **Am J Hum Genet** (1996), 59:45-51.

55. Macek M Jr., Mercier B, Mackova A, Miller PW, Hamosh A, Ferec A, and Cutting GR. Sensitivity of the denaturing gradient gel electrophoresis technique in detection of known mutations and novel Asian mutations in the CFTR gene. **Hum Mutat** (1997) 9:136-147.

56. Macek M Jr., Macek M Sr., Mackova A, Nash E, Hamosh A, Varon-Mateeva R, Reis A, Schmidtke J, Sperling K, Maestri NE, Krawczak M, and Cutting GR. Allele status of the CS.7/*Hha* I polymorphism 5' of the CFTR gene may be associated with female survival in normal Czech individuals. **Hum Genet** (1997), 99:565-572

57. Qian H, Hyatt G, Schanzer A, Hazra R, Hackam AS, Cutting, GR and Dowling JE. A comparison of $GABA_C$ and ρ subunit receptors from the white perch retina. **Vis Neurosci** (1997), 14:1-9.

58. Macek M Jr, Mackova A, Hamosh A, Hilman BC, Seldon RF, Lucotte G, Friedman KJ, Knowles MR, Rosenstein BJ and Cutting GR. Identification of Common CF Mutations in African-Americans With Cystic Fibrosis Increases the Detection Rate to 75%. **Am J Hum Genet** (1997), 60:1122-1127.

59. Hackam AS, Wang T-L, Guggino WB, and Cutting GR. The N-terminal domain of human GABA receptor rho-1 subunits contains signals for homooligomeric and heterooligomeric interaction. **J Biol Chem** (1997), 272:13750-13757.

60. Hackam AS, Wang T-L, Guggino WB and Cutting GR. A one hundred amino acid region in the GABA ρ1 subunit confers robust homooligomeric expression. **NeuroReport** (1997) 8:1425-1430.

61. Hamosh A, FitzSimmons SC, Macek M Jr., Knowles MR, Rosenstein BJ and Cutting GR. Comparison of the clinical manifestations of cystic fibrosis in black and white patients. **J Pediatrics** (1998), 132 255-259

62. Enz R and Cutting GR. Molecular composition of GABAC receptors. **Vision Res** (1998) 38 (10): 1431-1441.

63. Hackam AS, Wang T-L, Guggino WB and Cutting GR. The amino termini of GABA ρ and GABAA subunits contain sequences for their selective interaction in vitro. **J Neurochem** (1998), 70: 40-46

64. Mickle J, Macek M Jr, Fulmer-Smentek S, Egan E, Schweibert E, Guggino WB, Moss R, and Cutting GR. A mutation in the cystic fibrosis transmembrane conductance regulator associated with elevated sweat chloride concentrations in the absence of cystic fibrosis. **Hum Mol Genet** (1998), 7: 729-735.

65. Schwiebert EM, Cid-Soto LP, Stafford D, Carter M, Murray C, Guggino WB and Cutting GR. Analysis of ClC-2 channels as an alternative pathway for chloride conduction in cystic fibrosis airway cells. **Proc Natl Acad Sci USA** (1998), 95: 3879-3884.

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XIV. Inventions/Patents
Cystic Fibrosis Mutation Cluster
U.S. patent number: 5,407,796 Awarded : April 18,1995
Inventors: Cutting, G. R., Antonarakis, S.E. and Kazazian, Jr., H.H.